AN EVAULATION OF PATIENTS UNDERGOING LAPAROSCOPIC NISSENS FUNDOPLICATION

This dissertation is submitted to PSG Institute of Medical Sciences and Research in partial fulfillment of the regulations for the M.S (General Surgery) Degree Examination, April 2016

By

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CERTIFICATE

This is to certify that this dissertation entitled **"AN EVAULATION OF PATIENTS UNDERGOING LAPAROSCOPIC NISSENS FUNDOPLICATION"** is a record of bonafide research work done by **Dr.Prasanna Kumar S**, under my guidance and supervision in the Department of General Surgery, PSG Institute of Medical Sciences and Research, Coimbatore – 641004.

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DECLARATION

I, Dr. Prasanna Kumar S, solemnly declare that this dissertation "**AN EVAULATION OF PATIENTS UNDERGOING LAPAROSCOPIC NISSENS FUNDOPLICATION"** is a bonafide record of work done by me in the Department of General Surgery, PSG institute of Medical Sciences & Research, Coimbatore, under the guidance of Dr.Balashanmugam.T.S, Professor of Surgery. This dissertation is submitted to The Tamilnadu Dr.M.G.R. Medical University, Chennai, in partial fulfilment of the University regulations for the award of MS Degree (General Surgery) Branch-I, Examination to be held in April 2016.

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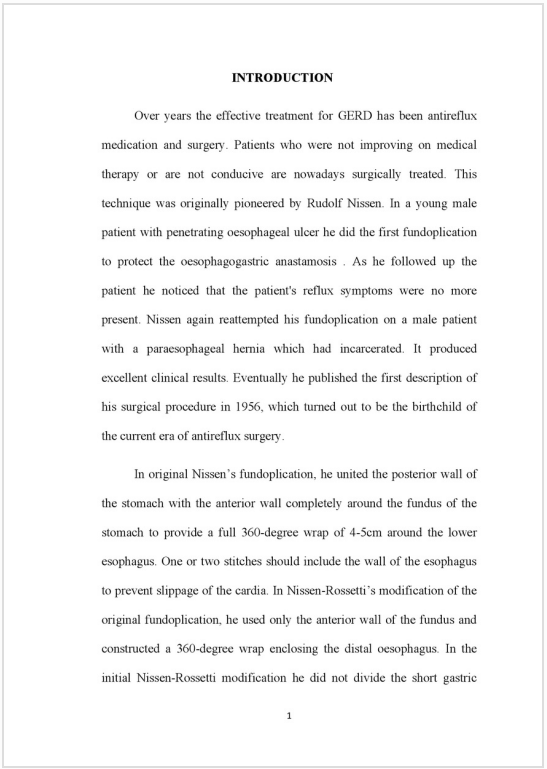

INTRODUCTION

Over years the effective treatment for GERD has been antireflux medication and surgery. Patients who were not improving on medical therapy or are not conducive are nowadays surgically treated. This technique was originally pioneered by Rudolf Nissen. In a young male patient with penetrating oesophageal ulcer he did the first fundoplication to protect the oesophagogastric anastamosis . As he followed up the patient he noticed that the patient's reflux symptoms were no more present. Nissen again reattempted his fundoplication on a male patient with a paraesophageal hernia which had incarcerated. It produced excellent clinical results. Eventually he published the first description of his surgical procedure in 1956, which turned out to be the birthchild of the current era of antireflux surgery.

In original Nissen's fundoplication, he united the posterior wall of the stomach with the anterior wall completely around the fundus of the stomach to provide a full 360-degree wrap of 4-5cm around the lower esophagus. One or two stitches should include the wall of the esophagus to prevent slippage of the cardia. In Nissen-Rossetti's modification of the original fundoplication, he used only the anterior wall of the fundus and constructed a 360-degree wrap enclosing the distal oesophagus. In the initial Nissen-Rossetti modification he did not divide the short gastric

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vessels. However, when a tension-free wrap could not be obtained, the short gastric vessels were sacrificed. The role of Operative treatment for Gastro Oesophageal Reflux Diseases and Hiatus Hernias have changed dramatically during the 90's. The driving force behind increased surgical numbers is the development of minimal invasive surgery. [3,4,5]

Although the techniques of Antireflux surgery has not changed, the approach has become more acceptable to the patient and referring physician because of small incisions, relatively short hospital stay and lack of post operative pain when compared to open approach.

AIM OF THE STUDY

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To assess whether Laparoscopic Nissen's floppy fundoplication really improves the quality of life compared to medical management and to assess the usefulness of surgery based on risk vs benefit ratio To evaluate the patients undergoing Laparoscopic Nissens fundoplication by comparison of their preoperative and postoperative Quality of life, dependency on medication and endoscopic findings.

REVIEW OF LITERATURE

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HISTORY :

It was not until early 1900 till radiological studies gained importance that hiatus hernia was recognized to be a pathologic entity. In mid 1930's post world war 1 it was identified as the precipitating cause for Oesophagitis. Initially the symptoms of Gastro oesophageal reflux was associated with hiatus hernia. This led to conclusion of hernia being the primary cause and attempts were made to correct hernia with simple closure of crura. However since lower oesophageal sphincter was not repaired, this procedure was a uniform failure and never gained universal acceptance.

In 1951, Philip Allison and Norman Barrett established the relationship between hiatal hernia, gastrooesophageal reflux and erosive oesophagitis. Allison explained that the crural sling and clasp musculature is the anatomic correlate to the Lower Oesophageal Sphincter and described it to be the primary mechanism which helps in preventing pathologic reflux. He performed a procedure where he restored the antireflux mechanism by reducing the cardia of stomach

which was herniating, by suturing to the diaphragmatic abdominal surface and lastly by loose closure of the hiatus.

However, this technique did not produce the desired results and his long-term recurrence rate of hiatus hernia at end of 20 years was almost 49%. Still, his repair represented the first effort in the right direction and he was the first to emphasize the importance of placing Gastro oesophageal junction in the intra abdominal position. He was thus credited to have initiated the modern era of antireflux surgery.

Subsequent efforts were made to develop procedures which anchored the lower oesophagus in the intra abdominal position. Initially, the surgeries employed were various forms of Gastropexy, where the stomach was pulled down in the abdomen and attached to the anterior abdominal wall or any posterior peritoneal structure strong enough to maintain it. This however placed the stomach and oesophagus under a great deal of continual tension which further stressed respiration and swallowing.

This was followed by Hill's procedure where gastro oesophageal junction was anchored posteriorly to the median arcuate ligament. Since this also did not produce the desired results, Belsey Mark 4 and Nissens fundoplication came into vogue. Belsey mark 4 is in essence a partial

270-280° fundoplication whereas Nissens is complete 360° fundoplication.

In 1939, Rudolf Nissen did a fundoplication on a male patient with penetrating oesophageal ulcer to safeguard his oesophagogastric anastomosis. During regular post operative revisits, he noted complete disappearance of his reflux symptoms. After few years, fed up with the then conventional hiatus hernia surgeries, Nissen again did his fundoplication on a male patient, this time with paraoesophageal hernia which had gone for incarceration. He had excellent outcomes. Nissen first published the description of his surgery in 1956, thereby pioneering the modern era of antireflux surgery. However Nissens fundoplication also had severe post operative complications, most of which could be attributed to faulty techniques and inaccurate patient selection. It was not a simple matter of wrapping the stomach around the lower oesophagus. Judgement and experience is vital in determining how long and how tight to make the fundoplication, what portion of the stomach should be used and what all conditions preclude the use of this surgery. If the fundoplication is too long or tight, it can result in dysphagia / odynophagia. Surgeons started introducing varieties of partial fundoplication to get around this problem. These included Dor (Anterior 180), Toupet (Posterior 270) and Belsey (270 Anterior Transthoracic).

The problems lie in suturing the fundus of stomach to the oesophagus. Besides subjecting the suture line to a great deal of stress, it also has limited durability. This high degree of disruption never allowed these procedures to take off.

The most durable of these partial fundoplication techniques was the Belsey Mark 4 operation. In this procedure which is performed transthoracic the oesophagus is adequately mobilized to avoid tension. In situations where the oesophagus has shortened, aggressive mobilization does not produce a tension free repair. To counter this, oesophageal lengthening technique was done by making a tube about the diameter of the oesophagus and 5cm in length in the lesser curvature of the stomach and constructing a Belsey fundoplication around the tube. This was called the Collin Belsey repair named after Dr. Leigh Collin and Dr. Ronald Belsey. The major drawback of this procedure is acid production within the tubularised portion of the stomach. Also this procedure is difficult to teach and has very less margin for error. In an experienced surgeon Mark Belsey operation has similar success to Nissens fundoplication.

The role for surgery as a treatment for Gastro Oesophageal Reflux Disease and Hiatal Hernias changed dramatically during the 90’s post the laparoscopic era.

Once a not so common procedure, nowadays many antireflux surgeries and hiatal hernia repairs are being performed in increased number of hospitals around the world, the driving force behind increased surgical numbers being the advancement of minimal invasive surgery.

Although the basic technique of Nissens original fundoplication has not changed, the approach has become far more acceptable to both the patient and referring doctor because of smaller incisions, relatively shorter duration of hospital stay and minimal post operative pain when compared to the open technique.

PATHOPHYSIOLOGY

The oesophagus, lower oesophageal sphincter and stomach may be viewed similar to a plumbing circuit, as put forward by Stein and co. He compared functioning of the oesophagus with an antegrade pump, the Lower Oesophageal Sphincter to a valve and stomach to that of a reservoir. The abnormalities which cause GERD may arise from any of the above three.

Poor motility of the oesophagus causes reduced clearing of the acid. Dysfunction of LES leads to reflux of huge quantities of stomach acid secretion. Delay in emptying of the stomach leads to increasing

volume and pressure within the stomach, ultimately defeating the valve mechanism causing GERD.

Both medically and surgically it is important in identifying the defective components to give proper efficacious treatment.

Oesophageal defense mechanisms :

Oesophageal defense mechanisms are broadly classified into 2 categories

1. Oesophageal clearance mechanism and
2. Mucosal resistance

Proper oesophageal clearance becomes extremely important in preventing injury to the mucosa. Oesophageal clearance should be adequate enough to neutralize acid reflux through the lower oesophageal sphincter.

Mechanical clearance is achieved through oesophageal peristalsis, whereas chemical clearance is achieved with saliva. In normal clearance, the time of exposure of the esophagus to refluxed acid or bile and gastric acid mixture is limited, whereas in case of abnormality in peristalsis there is inefficiency and delay in clearing the acid.

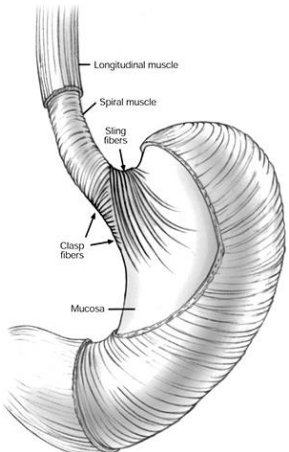
It is still not clear whether either the primary defect or increased oesophageal acid exposure is the cause of peristaltic dysfunction.

Dysfunction of LES

The lower oesophageal sphincter (LES) is a high pressure zone with raised intraluminal pressures at gastro oesophageal junction as defined by manometry. It is not distinct as an anatomical structure but as a unique physiological entity located just cephalad to the Gastro oesophageal junction.

Several factors contribute to this high pressure zone :

1. **Intrinsic musculature** of the distal oesophagus. They remain in a state of tonic contraction. They relax during swallowing and return to the contraction state post swallowing.
2. **Sling fibres of Cardia** These fibres are at the same anatomical depth as the circular fibres of the oesophagus. However their orientation is in a different direction. They run from the fundus of Cardia to the lesser curvature diagonally. These fibres are responsible for the lower oesophageal high pressure zone.



3. **Diaphragm** - During inspiration the antero posterior diameter of the crural opening is decreased causing an increase in pressure in the lower oesophagus. Usually LES pressure is assessed at mid or end expiration

4. **Transmitted pressure of abdominal cavity** – Intra abdominal pressure is usually higher than intra thoracic pressure which is negative. A Gastro Oesophageal Junction firmly anchored within the abdominal cavity will be exposed to greater transmural pressure than one which herniates into the posterior mediastinum

For proper functioning of Lower Oesophageal Sphincter, this junction must be located intra abdominally so as to facilitate the crura of the diaphragm to help the extrinsic sphincteric function of LES. Besides, the LES should be of normal length with normal sphincter pressure and should have normal regular number of transient relaxation episodes (relaxing of LES when there is no swallowing).

Lower Oesophageal Sphincter dysfunction is caused by any of the above mechanisms:

Most common being in the below order,

- Transient relaxed LES
- Permanently relaxed LES (and)
- Transient increase of intra-abdominal pressure to overcome lower oesophageal sphincter pressure.

Gastro Oesophageal Reflux occurs when the high pressure zone in lower oesophagus is inadequate to prevent reflux of gastric contents or when a sphincter with normal pressure spontaneously relaxes and is dissociated with the peristaltic wave of the body of oesophagus.

Even small changes in high pressure zone can compromise the effectiveness of LES. Thus reflux occurs in normal people too.

Delayed gastric emptying

The postulated theory of delayed stomach emptying leading to Gastrooesophageal reflux is due to increasing reflux of gastric contents leading to increase in pressure within the stomach leading to increase in the pressure pressing on the LES. LES pressure is ultimately overwhelmed leading to gastric reflux.

Hiatus hernia

Hiatal hernia is frequent in patients suffering from reflux disease. However there can exist patients with Hiatus hernia not having any reflux symptoms.

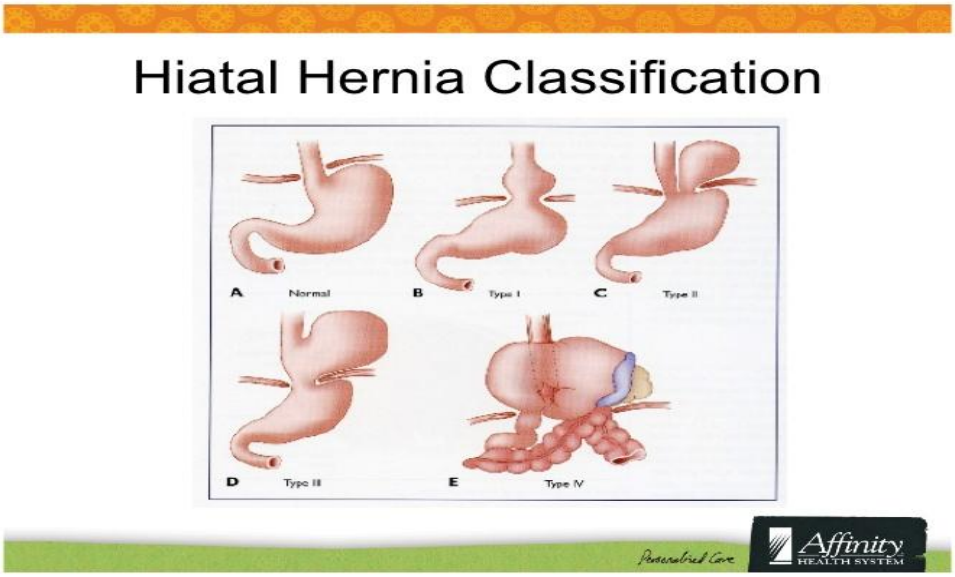
Types of Hiatal Hernias

Type 1 – Sliding hiatal hernia – (90%) When Gasto Oesophageal Junction is not maintained intraabdominally by phrenooesophageal ligament and herniates into mediastinum. (phrenooesophageal membrane continues with endoabdominal fascia which reflects onto the oesophagus at hiatus just superficial to the peritoneal reflection at hiatus continuing into the mediastinum)

Type 2 Rolling hernias (Paraoesophageal hernia) – when GEJ is anchored in abdomen but hiatal defect is large enough to allow herniation of viscera into the mediastinum. Also playing a role is the negative intrathoracic pressure which also aids this migration.

Type 3 Mixed hernias (combination of both the above)

Type 4 – when there is herniation of Colon and Spleen



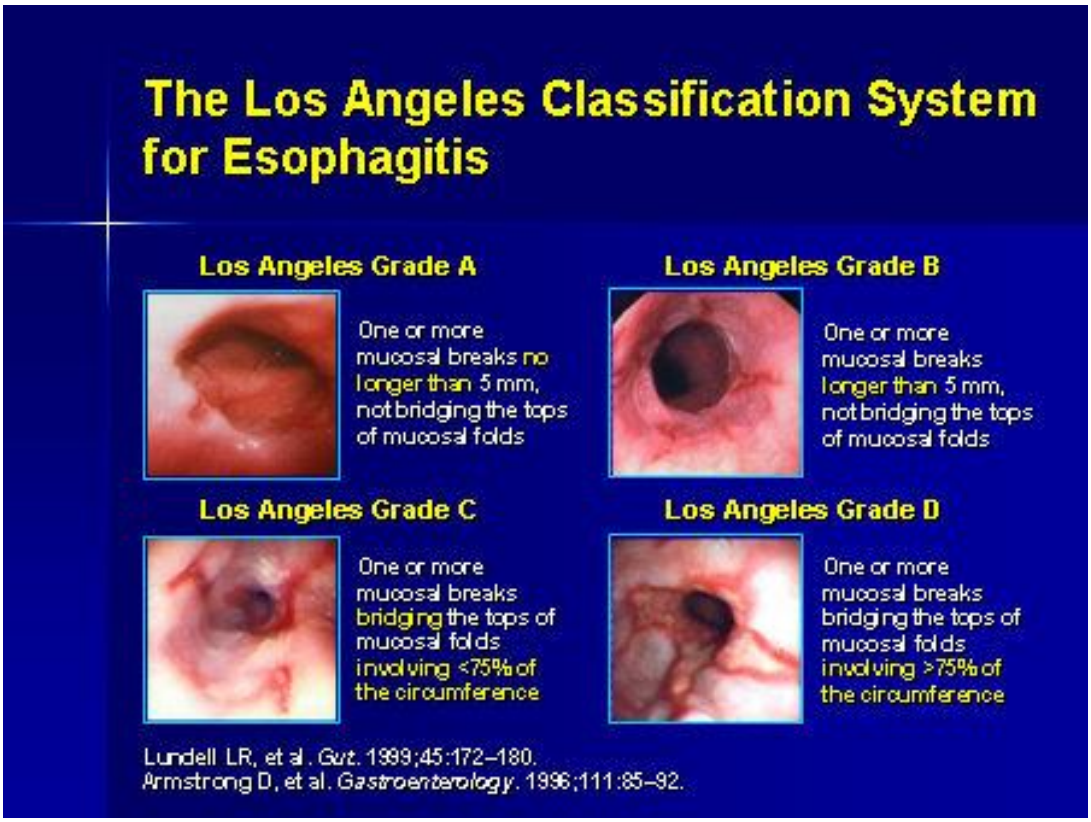
Obesity as a contributing factor

Studies have revealed GERD to be more common in patients with morbid obesity and those with high Body Mass Index. There is a hypothesis that states, the more obese the patient, the increased exposure to oesophageal acid. Aiding this hypothesis is a documentation of dose-response association where an increase in BMI increases the incidence of gastrooesophageal reflux with its complications. Hence, the pathophysiology of GERD in morbidly obese patients is different from that of normal patients. The implication of this is that the correction of reflux in morbidly obese patients is better achieved with a procedure that first controls obesity.

GRADES OF GERD :

Los Angeles Classification

Grade A	Single mucosal breaks < 5 mm in maximal length
Grade B	One or more mucosal breaks > 5mm, but without continuity across mucosal folds
Grade C	Mucosal breaks continuous between > 2 mucosal folds, but involving less than 75% of the oesophageal circumference
Grade D	Mucosal breaks involving more than 75% of oesophageal circumference



B) The Savary – Miller grading system

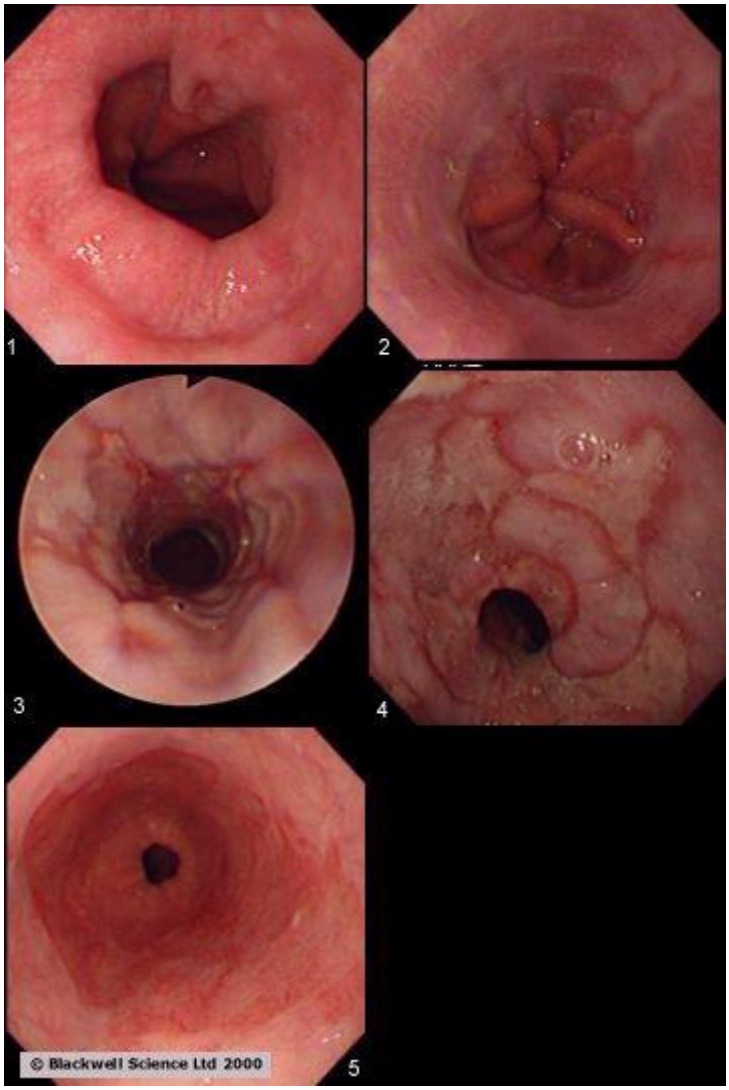
Grade 1: Single or multiple erosions on a single fold. Erosions may be exudative or erythematous.

Grade 2: Multiple erosions affecting multiple folds. Erosions may be confluent.

Grade 3: Multiple circumferential erosions.

Grade 4: Ulcer, stenosis or oesophageal shortening.

Grade 5: Barrett's epithelium. Columnar metaplasia in the form of circular or non-circular (islands or tongues) extensions.



COMPLICATIONS OF GERD :

Complications of GERD are due to injury to injury to oesophageal mucosa, Laryngeal or respiratory epithelial lining due to gastric acid

They are divided into :

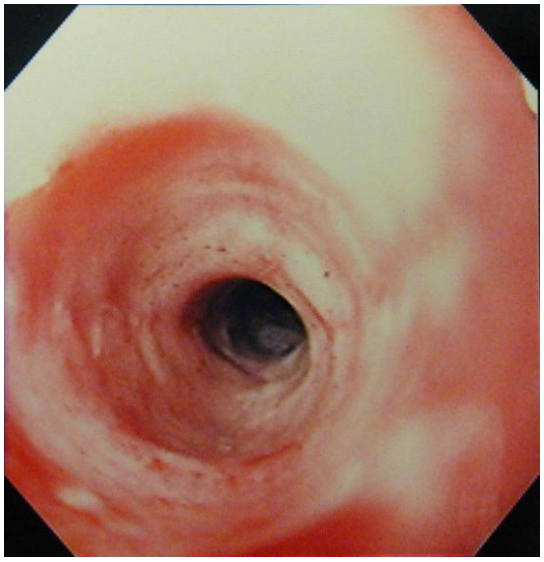
1. Mucosal complications – Oesophagitis, stricture
2. Extraoesophageal or respiratory complications – Laryngitis, recurrent pneumonia and progressive pulmonary fibrosis
3. Metaplastic and neoplastic complications – Barrett's oesophagus, Oesophageal adenocarcinoma

Mucosal complications :

The potential injurious components refluxing into oesophagus are due to gastric secretions like Hcl, pepsin, secretions from pancreas and biliary tract regurgitating from duodenum to stomach. Acid secretion by itself only minimally injures the gastric mucosa, but on combining with acid and pepsin it becomes very dangerous. H+ ions damage oesophageal mucosa only below pH of 2.

Similarly duodenal juice alone is not dangerous but combination of duodenal juice with gastric acid is highly deleterious.

Oesophageal stricture :



Diseases producing oesophageal stricture is classified into 3 groups

(1) Intrinsic diseases – include those which cause narrowing of oesophageal lumen by fibrosis, inflammatory or neoplastic changes.

(2) Extrinsic disease compressing upon oesophagus causing luminal narrowing by direct invasion or enlarged lymphadenopathy

(3) Diseases disrupting oesophageal peristalsis and normal functioning of LES by effect on oesophageal smooth muscle and its nerve supply.

Other causes for oesophageal stricture include

Acid peptic disease

Autoimmune disease

Infectious diseases

Iatrogenic cause

Caustic materials

Medication / radiation-induced

Malignancy

Oesophageal strictures are sequelae of Reflux resulting in oesophagitis. They take origin from the squamocolumnar junction averaging a length of 1-4 cms.

Two main causes of peptic oesophageal stricture are :

- **LES Dysfunction:** The LES mean pressure among patients having stricture is lower than those of healthy individuals. A study by Ahtaridis et al revealed a mean sphincter pressure of 4.9 mm Hg in patients having peptic oesophageal stricture in comparison with 20 mm Hg in normal control patients. Any Lower Oesophageal Sphincter pressure measuring lesser than 8 mm Hg shows significant correlation with presence of peptic oesophageal stricture.

- **Motility disorders:** They result in poor oesophageal clearance. It is found that 63% of patients having stricture suffered from other motility problems in comparison to 32% in case of normal subjects.

- Patients suffering from strictures complain of heartburn, dysphagia, odynophagia, impaction of food, loss of weight and chest pain.
- Progressive dysphagia from liquids to solids
- Atypical presentation including chronic cough, asthma, asthma due to aspiration of acid or food particles.

Investigations:

- **Barium oesophagram :** 100% sensitive in cases where diameter of lumen is smaller than 9 mm. It is 90% sensitive when diameter is larger than 10 mm.
- **Chest X ray PA and lateral :** Chest X Ray can be used as an adjunctive investigation when oesophageal stricture is suspected due to any extrinsic compression.
- **CT scan :** Has **60-68%** accuracy in its ability to estimate the depth of invasion of tumour. It has **82%** accuracy to help determine other organ invasion.

- **Endoscopic ultrasound (EUS) :** It has maximum accuracy in helping to identify the local invasion of tumour with its extent in case of any malignancy.

  It is 92% accurate in helping to estimate the depth of tumor invasion

Twenty-four hour oesophageal pH monitoring

Oesophageal manometry

Oesophagogastroduodenoscopy (EGD)

Treatment :

Medical – PPI's remain mainstay of management

Surgical - **Wire-guided polyvinyl bougies** - Savary-Gilliard, American dilators,

Fluoroscopic balloon dilators (FBD)

Intralesional injection of steroid

Expandable polyester silicone-covered stent

Oesophagus-sparing surgeries - Antireflux surgeries(Nissen's or Belsey partial fundoplication), Oesophageal lengthening by antireflux operations (Collin-Nissen or Belsey gastroplasty)

Oesophageal resection with reconstruction using gastric or colonic interpositioning or with jejunal segment

Respiratory complications :

Two mechanisms have been proposed.

1. **Reflux theory :** Respiratory symptoms are due to aspiration of gastric contents.

There are 5 points supporting reflux mechanism: 1.Evidences suggest a strong association between hiatus hernia and idiopathic pulmonary fibrosis 2.Pathological acid exposure in proximal oesophagus is often identified in these patients with respiratory symptoms and reflux 3.Scintigraphy studies show ingested Radio-isotope to be aspirated in patients having respiratory symptoms with reflux 4.Tracheal and oesophageal pH monitoring in patients with reflux show acidification of trachea associated with oesophageal acidification 5.Animal experiments reveal that HCL when ingested in trachea profoundly increases airway resistance

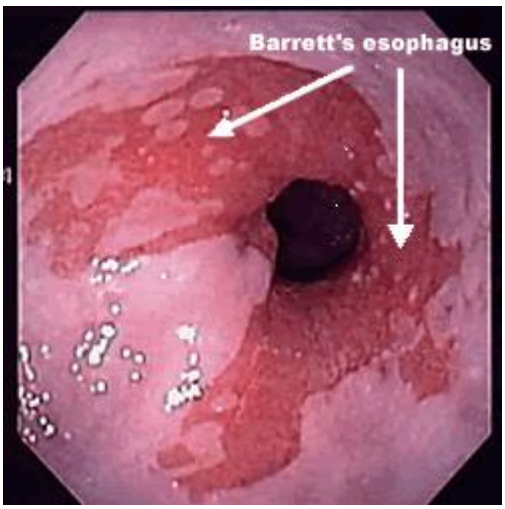
2. **Reflex theory:**

Vagal mediated bronchoconstriction follows acidification in lower oesophagus. In support of this theory bronchoconstriction occurs after infusing acid into lower oesophagus. This is due to common embryonal

origin of tracheooesophageal tract and its shared nerve supply from vagus. Treatment is by Proton pump inhibitor or Antireflux surgery

Metaplasia and Neoplasia :

Barret's oesophagus :



It presents as an entity where the tubular oesophageal lining of squamous epithelium is replaced by columnar epithelium. Norman Barrett first described it in 1950. It is an acquired abnormality with an incidence of 7-10% in patients suffering from GERD and it signifies the end stage of the natural history of the disease. It is identified when the columnar mucosa extends atleast 3cm into the oesophagus or during endoscopy by intestinal metaplasia of any length being visible. The hallmark of intestinal metaplasia is the presence of Goblet cells.

Pathophysiology : Metaplasia at Gastro oesophageal junction begins with distal oesophageal squamous mucosa converting to

cardiac type epithelium. This is because of the exposure to stomach acids and contents into the oesophagus resulting from prolapse of oesophageal squamous mucosa. This causes inflammation in the Gastro oesophageal junction and metaplasia both of which results in muscle function loss and defective LES, permiting free reflux with increased risk of of mucosal injury. Intestinal metaplasia within the LES may result in Barrett's metaplasia of the body of oesophagus.

Treatment :

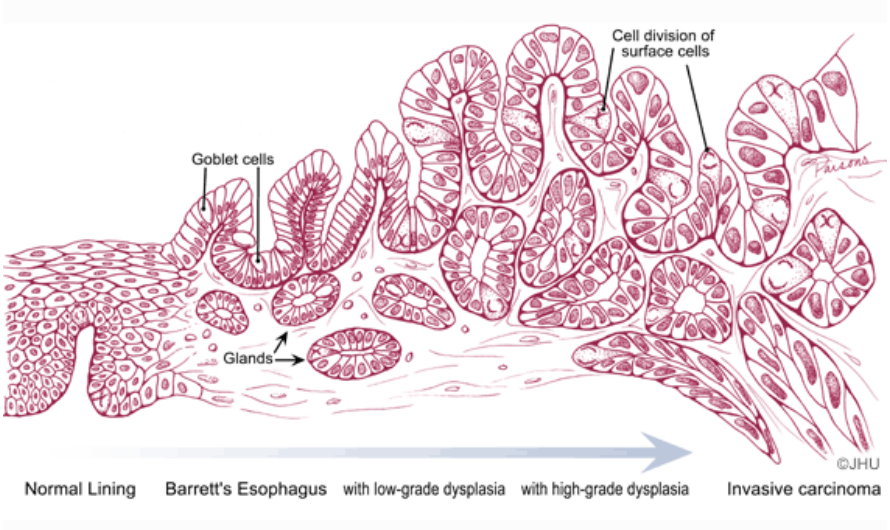
Based on classification of Barrett's oesophagus

1. **No dysplasia**
2. **Indefinite for dysplasia –** Aggressive antireflux therapy (60mg/day PPI), Repeat biopsy in 3 months
3. **Low grade dysplasia** – Aggressive antireflux therapy. Sugical - Nissens fundoplication in medical refractory cases
4. **High grade dysplasia** – Confirmation by 2 experienced pathologists. Oesophagectomy (?extent)

An important consideration in Barrett's oesophagus is, these patients present with severe Gastro oesophageal reflux, and its sequelae, like large Hiatus hernia, short oesophagus, stricture and poor motility. These factors make successful antireflux surgery a challenge. Data also suggests that

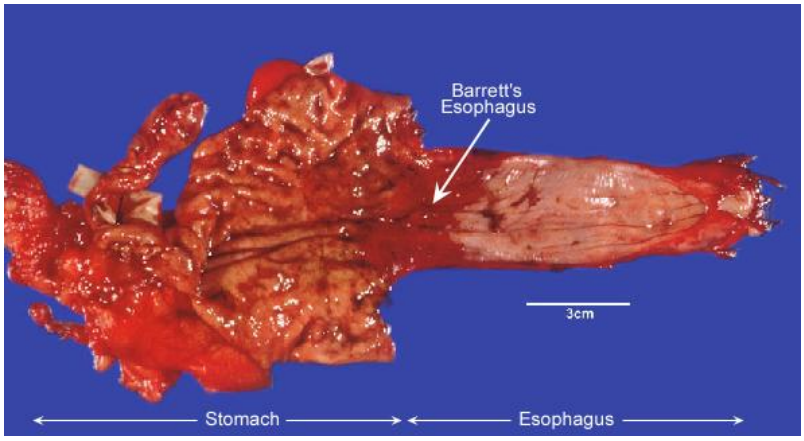
fundoplication in Barrett's oesophagus do not have the same level of success as those performed in patients without Barrett's oesophagus

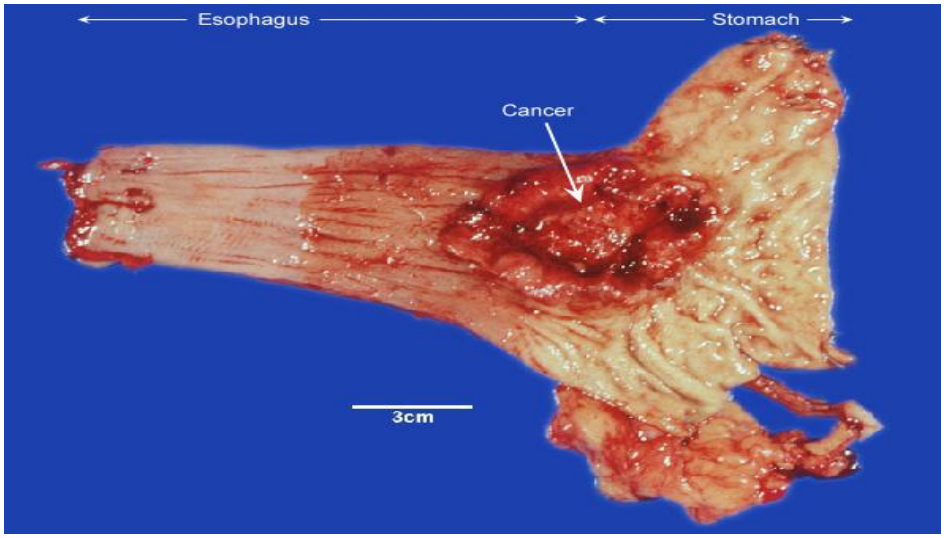
Barrett's Oesophagus and adenocarcinoma :



Barrett's esophagus is a premalignant condition. Its malignant sequela, oesophagogastric junctional adenocarcinoma, has a mortality rate of over 85%. The relative risk of esophageal adenocarcinoma is approximately 10 in those with Barret's esophagus, compared to the general population. Most patients with oesophageal carcinoma survive less than 1 year. In addition, Barrett's oesophagus has been reported in patients with no reflux symptoms. The risk of oesophageal adenocarcinoma is increased 30 to 40 times among patients with Barrett's compared with those without this condition. EAC continues to increase at a rate greater than any other cancer in the Western world (>500% since the 1970s), exceeding that of other more common cancers such as breast,

colon, lung, and prostate cancer. In 2009, it is estimated that 16,470 new cases of oesophageal cancer will be diagnosed in the United States, of which close to 60% will be adenocarcinomas. Despite all the recent advances in the diagnosis and management of this lethal cancer, the overall 5-year survival rate remains dismal





This specimen in above picture shows a carcinomatous lesion at the junction between the oesophagus and stomach. It is a large irregular mass. The main aim of endoscopy in Barrett's oesophagus lies in detecting these lesions early on when rate of cure is high

Approximately 30% of oesophageal cancers, who have undergone pre op chemo and radiotherapy present with no residual cancerous lesion in the resected specimen. These patients survive longer than surgery-alone patients. The treatment trials should be tailored to each patient to provide maximum benefit.

Treatment of choice for early oesophageal cancer proven on biopsy is surgical removal of intrathoracic oesophagus. Oesophageal adenocarcinomas can metastasize to chest lymph nodes. It is best to confirm the diagnosis before surgical resection.

In early detection of adenocarcinoma, in the presymptomatic stage in patients with Barrett's oesophagus, chances of a surgical cure is high - 50 to 80%.

The treatment of choice for patients with invasive oesophageal cancer is pre-op chemo RT followed by surgery. Each patient is staged using diagnostic modalities like CT scan, MRI, endoscopic ultrasound, and laparoscopic examination prior to chemo RT.

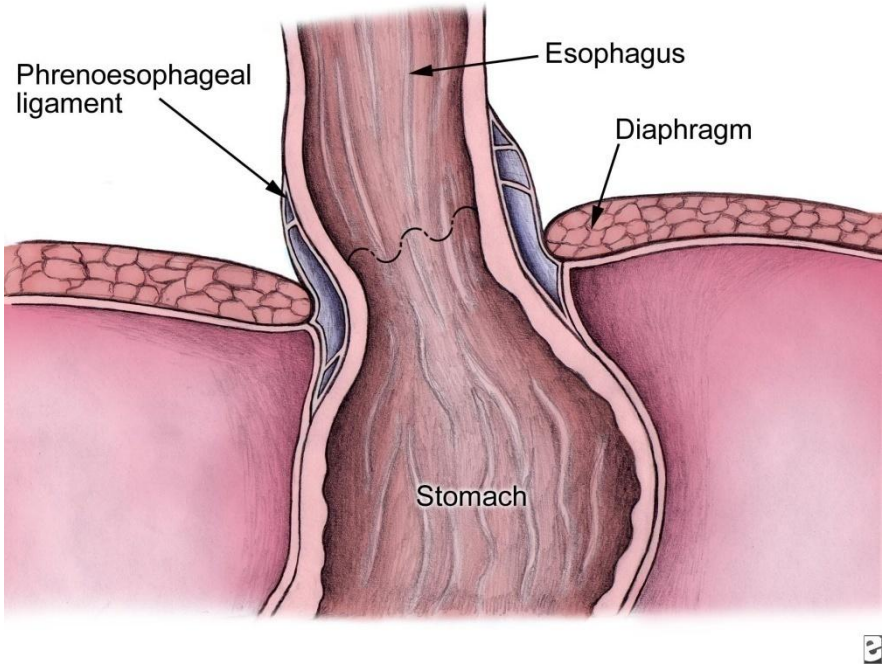
ANATOMY

The oesophagus can further be sub-divided into 1. Cervical oesophagus 2. Thoracic oesophagus and 3. Abdominal oesophagus. It

consists of an inner circular muscle layer and outer longitudinal muscle layer. There is a smooth transition of the upper proximal 1/3rd which comprises of striated muscle, to smooth muscle in the distal 2/3rd. The upper oesophagus comprises of upper oesophageal sphincter (UES), which has 2 muscles, Cricopharyngeus and Thyropharyngeus.

The distal lower thoracic oesophagus lies to the left of the midline. It continues to become the abdominal oesophagus in the region where thoracic oesophagus enters abdomen through the oesophageal hiatus. The oesophageal hiatus is formed from the Right crus of the diaphragm forming an oesophageal sling surrounding it with left and right pillars. The contraction of diaphragm causes narrowing of the oesophagus.

Relationship between phrenooesophageal ligament with oesophagus and diaphragm.



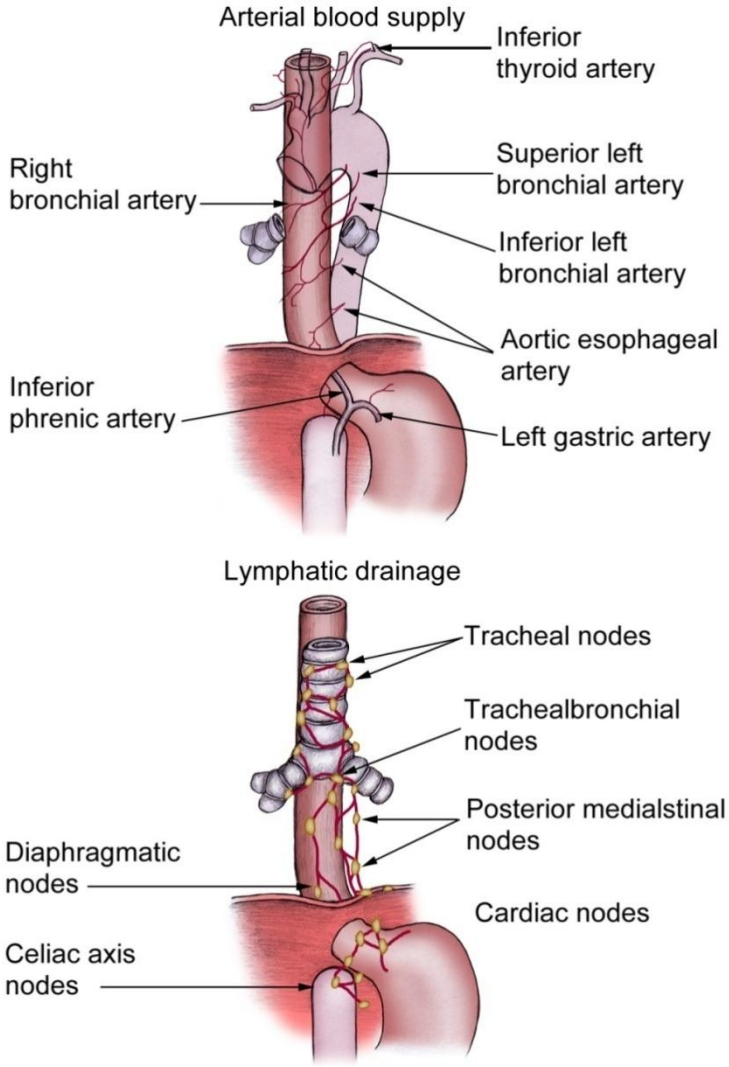
At this level, phrenooesophageal ligament encircles the oesophagus. The lower limit of this ligament is marked by a prominent pad of fat lying on the anterior oesophageal surface which demarcates the oesophagogastric junction. The junction is intraabdominal forming the His angle. This acute angle combined with normal abdominal oesophagus length causes normal closure of the oesophagus in case of high intra-gastric and intra-abdominal pressures.

The lower oesophageal sphincter or in other terms the distal oesophageal high-pressure zone (HPZ) comprises of the most distal segment of oesophagus measuring 2-5 cm length. Maintanence of adequate intra-abdominal HPZ is very important to prevent Gastro oesophageal reflux. This is formed by a complex architecture of smooth

muscle fibers, which are classically identified by oesophageal manometry. Gastro Oesophageal Reflux is due to malfunctioning or abnormalities of either one or more of these anatomic features. Hence a complete evaluation and correction pre operatively and intra operatively of all these defective anatomical features is necessary.

Blood supply of Oesophagus and Stomach

Oesophagus has segmental blood supply. Cervical oesophagus is supplied by inferior thyroid artery. Branches from bronchial arteries and directly off Aorta play a role in supplying the proximal oesophagus and distal thoracic oesophagus respectively. Left gastric artery branches and Inferior phrenic artery branches give supply to the abdominal oesophagus. A more consistent branch, Belsey artery plays a role in connecting the left gastric artery with inferior phrenic arteries.



Arterial blood supply and lymphatic drainage of the Oesophagus.

Stomach has a rich blood supply with overlapping vessels. The lesser curvature is supplied by left gastric artery from Coeliac trunk and Right gastric artery from Hepatic artery proper. The greater curvature derives supply from Right gastro epiploic originating from the gastroduodenal artery and Left gastroepiploic and Short gastric arteries originating from Splenic artery. Excellent collaterals around the stomach

allow ligation of many of the arteries around the stomach without any significant risk of ischemia.

Aberrant Left Hepatic Artery:

An aberrant left hepatic artery is a branching of the left gastric artery which provides arterial supply to Left lobe of liver. It passes through the gastrohepatic ligament to supply the left lobe. The incidence of this aberrant Left Hepatic artery ranges from 3% to 25%. Is is especially significant in surgeries for liver transplant and surgeries performed near the gastrohepatic ligament like oesophagogastrectomy, gastrectomy, gastric bypass, and antireflux procedures.

Although recent reports say that there is only transient liver enzyme elevation with Aberrant Left Hepatic Artery transection, reports confirming significant hepatic necrosis, even mortality are documented.

In presence of ALHA, dissection of hiatus during antireflux procedure provides more of a challenge. In laparoscopic Nissens fundoplication, preserving the ALHA is advocated and is accomplished with meticulous dissection and identification. Also according to recent reports, in adults safe division of this artery is necessary due to excessive bleeding and poses a potential risk of hepatic ischemia.

PHYSICAL EXAMINATION :

<u>TYPICAL OESOPHAGEAL SYMPTOMS</u> :

Heartburn (80%) – The commonest presenting symptom in GERD. Confined to epigastric and retrosternal areas. Can be felt as a caustic, stinging or burning sensation or discomfort, occurring generally post food intake, in supine position or bending over. There is no radiation of pain to the back.

Regurgitation (54%) – Effortless reflux of gastric and/or oesophageal contents into the pharynx. When gastric contents spill into the tracheobronchial tree it can cause respiratory complications. Regurgitation indicates progress of disease. Regurgitation comprises predominantly of digested food. Undigested food is indicative of achalasia / diverticulm

Predominantly **epigastric abdominal pain (29%)**

Dysphagia (23%) It is a sensation of food being stuck. It is more commonly felt retrosternally and is due to mechanical obstruction. Dysphagia is mostly for solids. It is an advanced symptom and can also be due to 1. primary oesophageal motility disorder 2. motility disorder

secondary to oesophagitis or 3.stricture formation. Equal intensity of Dysphagia for solids and liquids reveal a neuromuscular cause

Belching (15%)

Abdominal bloating (15%)

ATYPICAL OESOPHAGEAL SYMPTOMS :

Coughing & Wheezing are respiratory symptoms due to the aspiration of gastric contents into tracheobronchial tree. Approximately **50%** of patients with GERD-induced asthma do not experience heartburn.

Hoarseness (21%) is due to irritation of vocal cords by gastric reflux and is often experienced in the morning.

Aspiration (14%)

Globus (4 %)

Reflux is one of the most common causes of non cardiac chest pain. Reflux causing chest pain comprises of nearly 50% of the cases. The typical presentation of these patients is with pain resembling Myocardial infarction. Once a non cardiac cause of chest pain is ruled out, patient is started on a therapeutic high-dose of proton pump inhibitor (PPI's).

Symptom scoring for GERD :

GIQLI (Gastrointestinal quality of Life index):

GIQLI is graded from 0-144 points and five sub-items:

1. gastrointestinal symptoms
2. emotional status
3. physical functions
4. social functions
5. stress by medical treatment.

Scores for each sub item range between 0 and 4; Higher scores reflect better QoL.

Visick Classification of peptic ulcer surgical results :

Grade I	No symptoms
Grade II	Mild symptoms, Relieved by age
Grade IIIa	Symptoms relieved by care but patient satisfied with results
Grade IIIb	Symptoms not relieved by care and patient unhappy
Grade IV	No improvement

Modified Visick Classification :

Grade I No symptoms, perfect results

Grade II Patient states that results are perfect, but symptoms can be elicited

Grade III Mild to moderate symptoms, patient and surgeon satisfied with results

Grade IV Mild to moderate symptoms, patient and surgeon dissatisfied

WORKUP :

Mandatory workup include upper GI endoscopy.

Upper Gastrointestinal Endoscopy

Oesophagogastroduodenoscopy (EGD) is used to define the anatomy, identify the presence and also severity of complications of reflux disease - Oesophagitis, Barrett's oesophagus, Strictures. By taking the patient's history and pathologic analysis of biopsy specimens obtained with endoscopy, diagnosis of GERD can be made along with the grades and complications. Also anatomical problems like Hiatus hernia and its types can be identified.

Upper GI Endoscopy is as of now the most cost-effective diagnostic study to diagnose GERD.

The location of both crura of the diaphragm, gastro oesophageal junction and squamo columnar junction should always be recorded.

Non erosive oesophagitis is difficult to record endoscopically. Its presence can be confirmed by biopsy – showing mucosal infiltration with polymorphs, lymphocytes, eosinophils and balloon cells. The extension of relative height of mucosal papillae and hyperplasia of basal zone are further evidence of mucosal injury. These microscopic signs while providing corroborative evidence do not prove the presence of increased exposure to gastric juice as can occur from other forms of injury.

Endoscopically mucosal breaks defines oesophagitis according to Los Angeles classification.

Barretts oesophagus should be suspected when there is difficulty in visualizing the squamo columnar junction at its normal location and the appearance of redder more luxuriant mucosa than is normally seen in the lower oesophagus. Its presence is confirmatory of the reflux of gastric juice and must be confirmed with finding of columnar epithelium with intestinalization on microscopic inspection of biopsy. Multiple biopsies are taken cephalad to determine where Barrett's oesophagus ends and normal mucosa starts. Barrett's oesophagus can ulcerate, form stricture and has increased risk of malignancy and bleeding. The early signs of malignancy is dysplastic changes. Since they can occur randomly, we

should do a minimum of 4 biopsies for every 2cms. Biopsies are taken from the epithelial metaplasia. Due attention should be given to the squamo columnar junction where any mass, ulcer, nodules or inflammatory tissue should arouse suspicion of malignancy and warrants a biopsy.

Endoscopically hiatus hernia occurs as a pouch lined by folds of stomach mucosal ruggae which lies 2cm or greater above the crural margins of the diaphragm. A sliding hiatal hernia which is prominent is more often than not associated with increased gastro oesophageal reflux. In case of a paraoesophageal hernia, due care should be taken to exclude any gastric ulcer or gastritis within the pouch. The J maneuver helps evaluate the full circumference of the mucosal lining of the herniated stomach.

Oesophageal Manometry

Oesophageal manometry aids surgical planning by measuring the LES pressure. This helps to identify any motility disorders. It also helps in evaluating Oesophageal amplitudes and propagation of oesophageal swallows. Oesophageal manometry also helps in correctly positioning the probe if needed for 24-hour pH monitoring.

However, as per "**Guidelines for surgical treatment of gastroesophageal reflux disease",** done by Stefanidis D, Hope WW, Kohn GP *in Annals of Surg. Endoscopy Nov 2010 and* according to the study **"The preoperative evaluation of patients considered for laparoscopic antireflux surgery"** by Waring JP, Hunter JG, Oddsdottir M, Katz E, *Am J Gastroenterology*, **less than 10% of manometry findings alter surgical planning. Also, no literature supports mandatory preoperative manometry testing. Rather, manometry may be considered in patients who do not respond to empiric medical treatment and have normal endoscopy findings.** [6,7,9,10,11,50]

Ambulatory 24-Hour pH Monitoring

24-hour pH monitoring during rest and ambulation helps to establish diagnosis of Gastro Oesophageal Reflux. It has a high sensitivity of 96% and a specificity of 95% in patients when their history is not conclusive and in patients where atypical symptoms dominate the clinical picture, or in a case of Upper GI endoscopy showing no complications of reflux. It quantifies the gastrooesophageal reflux and allows a correlation between the symptoms of reflux and the episodes of reflux. Either a 24-hour ambulatory oesophageal pH-meter or the 48-hour wireless oesophageal pH-monitor probe is used.

However, while pH testing is considered the criterion standard for diagnosis of GERD, **routine use has found it to be only of marginal benefit**. [10,50] This test is best used in the **absence of endoscopic evidence of reflux or when the diagnosis is unclear** according to Frantzides CT, Carlson MA, Madan AK, Stewart ET, Smith C. in **"Selective use of esophageal manometry and 24-hour pH monitoring before laparoscopic fundoplication"** *Journal of American Coll Surg*. 2003 and "**Guidelines for surgical treatment of gastroesophageal reflux disease",** done by Stefanidis D, Hope WW, Kohn GP *in Annals of Surg. Endoscopy Nov 2010*. Thus, Patients with oesophagitis confirmed on endoscopy are not in need of pH monitoring for establishing a diagnosis of Gastro oesophageal reflux.[6,10,11]

To conclude, the following are indications for oesophageal manometry and pH monitoring over 24 hours which include:

- Persisting symptoms inspite of adequate anti secretory drugs such as PPI's and recurrence of symptoms post discontinuing medication **in the absence of endoscopic findings** and in investigating atypical symptoms like chest pain or asthma in subjects not having oesophagitis.

Also, as of present there is no role for CT, MRI or ultrasonography in routinely evaluating patients with reflux disease. [27,41,50]

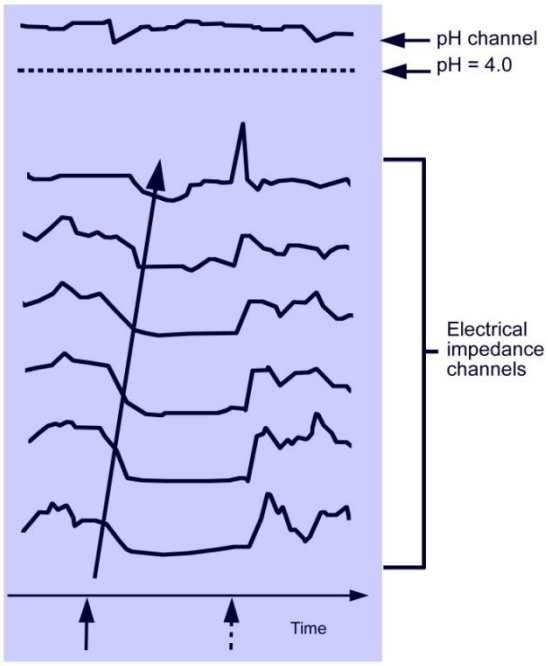
Imaging in GERD :

Plain radiography is not very useful to evaluate patients for Gastro oesophageal reflux, however it plays a role in evaluating lung status and basic anatomy. X ray chest can identify large hiatus hernias. It must be noted however that small hernias can be easily missed. Contrast-enhanced upper GI studies were the initial diagnostic radiologic test of choice in working up a patient with suspected GERD. Oesophageal inflammatory diseases and neoplasms are better detected with double-contrast study. However, single-contrast techniques still have more sensitivity in detecting structural anomalies like hiatus hernias, strictures and oesophageal rings.

Delay in gastric emptying time is observed in nearly 60% of the patients suffering from Gastro oesophageal reflux. Patients suffering from a delay in gastric emptying time classically have bloating post food intake and fullness of abdomen besides other symptoms. In these patients studies which determine Gastric-emptying will be useful to evaluate whether delayed gastric emptying is the contributor of GERD symptoms.

Intraluminal Oesophageal Electrical Impedance

Intra-luminal oesophageal electrical impedance (EEI) is a more recent test which detects both acidic and nonacidic reflux by measuring retrograde flow within the oesophagus. Gastrooesophageal reflux episodes which last as briefly as 15 seconds are measured



This image represents concomitant intraoesophageal pH and oesophageal electrical impedance measurement. The vertical solid arrow is used to indicate commencing of a nonacid gastro oesophageal reflux episode. The vertical dashed arrow indicates the onset of a normal swallow.

In adult studies, impedance measurements are used in conjunction with 24-hour intra oesophageal pH monitoring, to provide a more

complete picture of bolus movement within the oesophagus. EEI is not yet thoroughly validated and normal values for paediatric age group are yet to be determined.

APPROACH CONSIDERATIONS

Treatment of gastro oesophageal reflux disease is by a stepwise approach. The goal is to control symptoms, to heal the oesophagitis and to prevent recurrent oesophagitis or other complications.

The treatment is based on

(1) Lifestyle modification

(2) Control of gastric acid secretion through a. Medical therapy with antacids / PPIs or b. Surgical treatment with corrective antireflux surgery.

Approximately 80% of patients have recurrent but non progressive forms of GERD which can be controlled with medications alone. Identifying the other 20% of patients who have the progressive form is important, because these patients may develop severe complications in future such as strictures or Barrett oesophagus. For these patients surgical treatment should be given as an option in earlier stage itself to avoid the sequelae of the disease that can have far more serious consequences.

By use of a self management tool by patient, eg. a self-administered GERD Questionnaire (Gerd Q) for stratification of patients will help in improving GERD management of patients in primary health care setting.

Lifestyle Modification

- Weight loss especially in obese patients
- Avoid alcohol, chocolate, citrus juices and spicy food
- Taking small frequent meals
- To wait for atleast 3 hours after a meal before lying supine
- Elevate the head end of bed by 8 inches

Lifestyle modifications also constitute the first line of management in pregnant women with GERD. Patients are adviced elevation of head end of the bed; to avoid bending down or assuming a stooping positions; to consume small quantity meals with increased frequency and avoid intake of food(except maybe liquids) before 3 hours of bedtime.

Pharmacologic Therapy

1. Antacids

Antacids were the mainstay drugs for controlling mild symptoms of GERD in the 1970's and still remain effective. Antacids should be taken after each meal and before going to bed.

2. H2 receptor antagonists and H2 blockers

H2 receptor antagonists were first-line drugs in patients suffering from mild to moderate symptoms with Grade I and Grade II GERD. Options include Ranitidine, Cimetidine, Famotidine. However, they were found to be effective in curing only mild oesophagitis in nearly 80% of patients with Gastro oesophageal reflux and also in providing maintenance dose to help prevent relapse. Tachyphylaxis was also observed, leading on to suggest that pharmacological tolerance may develop which causes reduction in the efficiency of these drugs in the long run. The H2 receptor antagonists were reversible competitive histamine blockers at the H2 receptor sites, especially in the parietal cells of gastric mucosa, where they cause inhibition of acid secretion. Being highly selective, they have no effect on the H1 receptors

3. Proton Pump Inhibitors

Proton pump inhibitors (PPIs) belong to the class of medicines that are ubiquitous in the day to day practice of any gastroenterologist. This has replaced h2–receptor antagonists in treating moderate to severe stomach acid reflux and are the drugs of choice for prophylaxis of GI injury. Their safety among other pharmacological agents has been

undisputed. PPIs remain at the top among the list of widely sold drugs worldwide. The first PPI, Omeprazole, introduced in 1980's is in WHO's List of essential medicines.

They are used in treating many conditions like:

- Dyspepsia
- Peptic ulcer disease, which also includes after endoscopic treatment for bleeding
- For Helicobacter pylori eradication therapy
- GERD – in patients having symptoms but have no findings on endoscopy to suggest reflux disease
- Associated laryngo-pharyngeal reflux which causes chronic cough and laryngitis
- Barrett's oesophagus
- To treat Stress gastric ulcers and their prevention in critical care patients
- Gastrinoma and other conditions which may lead on to increased secretion of acid. This includes Zollinger–Ellison syndrome

Mechanism of Action:

Proton pump inhibitors act by irreversible blockage of H^+/K^+ ATPase, of the gastric parietal cells. This is the last stage in

secretion of gastric acid, thus it directly causes H^+ ion secretion into the lumen of the stomach, thus making it an easy target for inhibition of acid secretion.

Due to the decrease in acid secretion in the stomach, faster healing of duodenal ulcers are promoted and pain due to indigestion and heartburn are reduced. Decreased quantities of gastric acid (hypochlorhydria) impairs protein digestion and nutrient absorption, especially Vitamin B_{12} and Calcium.

PPIs when given in inactive form, which when neutrally charged(lipophilic) can readily cross cell membranes into intracellular compartment with acidic environment. Here the inactive drug is protonated to rearrange to its active form.

PPI"s include :

Omeprazole

Lansoprazole

Rabeprazole

Esomeprazole

Pantoprazole

Ilaprazole (not FDA approved as of October 2013)

Pharmacokinetics

The rate of omeprazole absorption can be decreased by concomitant intake of food. Also, absorption of lansoprazole and esomeprazole is also decreased and delayed by food intake. However, according to reports, these pharmacokinetics have no significant impact on its efficacy. PPIs have a short half-life in human plasma (60–90 min), However, since they covalently bind to pump, their half-life of gastric acid secretion, inhibition is much longer. Half-life at site of action is roughly 24 hours.

Effects on Vitamin and Mineral Absorption

Iron : Though PPI's can reduce non heme iron absorption and decrease iron pool replenishment, this effect has not been well demonstrated in clinical practice.

Calcium : Evidence to suggest PPI's cause increased risk osteoporotic-related fractures is not well proven. Various studies and data on bone density loss and osteoporotic fractures are not convincing to suggest discontinuing PPIs for appropriate indications at correct doses. Also contrary to what was initially suggested PPIs can actually increase bone density by impairing osteoclastic activity, which facilitates the dissolution of bone matrix and its subsequent resorption.

Vitamin B12: Gastric acid causes absorption of B_{12} by initiating its release from dietary protein, causing B_{12} to bind to R protein. This B_{12}–R protein complex breaks down in duodenum, causing B_{12} to be absorbed in terminal ileum. Since B_{12} absorption depends on stomach acid, theoretically, long-term PPI's can impair their absorptive ability.

Magnesium : Low levels of magnesium can occur in patients on PPI therapy which can reverse when switched to H2-receptor antagonists.

Studies reveal a correlation between PPI use and *Clostridium difficile* infection, causing FDA to include a warning label on PPI's. Also concerns have been raised about small intestinal bacterial overgrowth and spontaneous bacterial peritonitis in older people taking PPIs and in subjects with irritable bowel syndrome on PPIs. Long-term use of PPI is also associated with development of benign polyps from fundic glands. These are not carcinogenic and resolve on discontinuation of PPIs. Also use of PPIs can mask gastric cancers and other gastric disorders. Hence physicians should be aware when prescribing. PPI's have also been associated with microscopic colitis.

Cardiovascular :

Relationship between PPI's and cardiovascular events are not very clear. PPIs with Aspirin is commonly used in cardiovascular patients for

gastric protection where Aspirin is given for its antiplatelet action. Similarly it is also done in the case of Clopidogrel. One suggested mechanism for this corelation is, PPIs bind and inhibit dimethylargininase enzyme which degrades asymmetric dimethylarginine (ADMA) causing increasing ADMA levels and decreasing bioavailable nitric oxide levels. In January 2009, the FDA issued a recommendation against the combined use of clopidogrel and all PPIs, subsequently revising their statement to recommend against potent CYP2C19 inhibitors, naming omeprazole, esomeprazole, and cimetidine. This recommendation was based on several high-profile retrospective database evaluations that found higher cardiac event rates (stent thrombosis, myocardial infarct, and death) in patients who were taking clopidogrel with PPI's than those on clopidogrel alone.

In general, proton pump inhibitors are well tolerated, and the incidence of short-term adverse effects is relatively low. Long-term use of PPIs has been less studied than short-term use, and the lack of data makes it difficult to issue definitive statements.

Other common adverse effects include headache, nausea, diarrhea, abdominal pain, fatigue, and dizziness. Infrequent adverse effects include rash, itch, flatulence, constipation, anxiety,

and depression. Also rarely, PPI use may be associated with occurrence of myopathies, including the serious reaction rhabdomyolysis.

4. **Prokinetics**

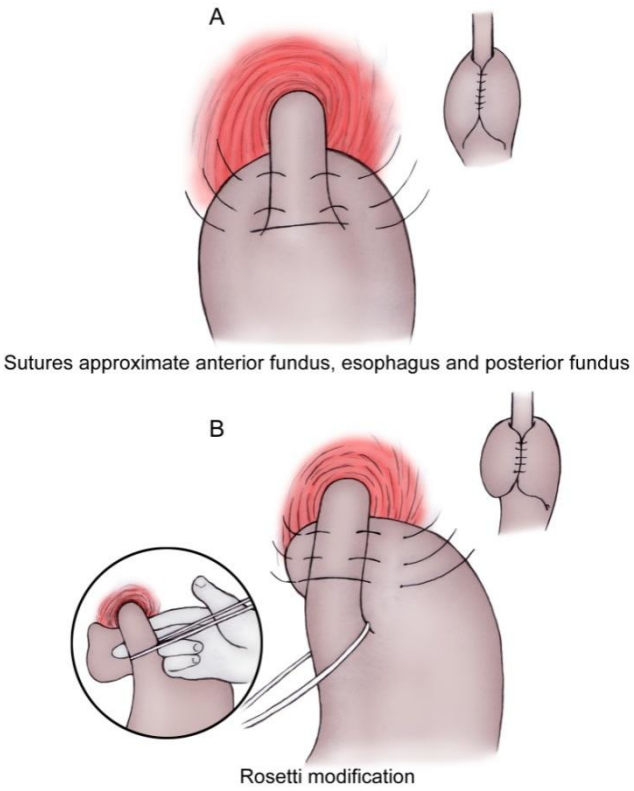
Prokinetics improve oesophageal and stomach motility. It also increases LES pressure thereby helping to reduce gastric reflux. They also accelerate gastric emptying. Prokinetic agents are mostly effective only in patients with mild symptoms. Those with more severe symptoms need additional acid-suppressing medication such as PPIs. Long-term prokinetic agent intake should be discouraged as they have serious, even potentially fatal complications. Metoclopramide is a Gastrointestinal prokinetic agent which causes an increase in GI motility, increasing resting oesophageal sphincter tone and relaxing the pyloric sphincter.

INDICATIONS FOR SURGICAL MANAGEMENT

Surgical therapy for gastrooesophageal reflux today is both by transthoracic and transabdominal fundoplications, using either partial (anterior or posterior) or circumferential wraps.

The commonest surgery performed today, both in children and adults is the Nissen's floppy 360° fundoplication, by transabdominal approach. First report of this procedure is in 1991, which was generally

well accepted and studied in adults, now Laparoscopic fundoplication is also gaining acceptance for use in children.



Indications for fundoplication :

- Patients with reflux symptoms not completely controlled by PPI's. It is also considered in patients with GERD under control who desire one-time definitive treatment.
- Barrett's oesophagus is also an indication for surgery (whether suppression of acid improves the outcome or prevents progression of Barrett's oesophagus remains unknown, but most authorities recommend complete acid suppression in patients with histologically proven Barrett's oesophagus)

- In patients with extraoesophageal GERD manifestations like(1) respiratory manifestations (eg, aspiration, cough, wheezing,) (2) ENT manifestations (eg, sore throat, otitis media, hoarseness of voice) and (3) dental manifestations (eg, erosion of enamel)
- Patients who are not compliant with medication
- Postmenopausal women with osteoporosis
- Patients having cardiac conduction defects
- Patients who cannot afford the long term costs of medical therapy

There have been several RCT's over the years which have challenged whether surgery benefits over medical management in controlling GERD.

According to a trial done by Spechler at the end of 10 years post fundoplication 62% of patients were on antireflux medications.[24]

Another randomized study by Anvari et al again emphasised the importance of surgery in treating GERD. They have proved that at the end of 1 year, the outcomes and symptoms among the surgical group was better than that kept on long term medical management. [17,18]

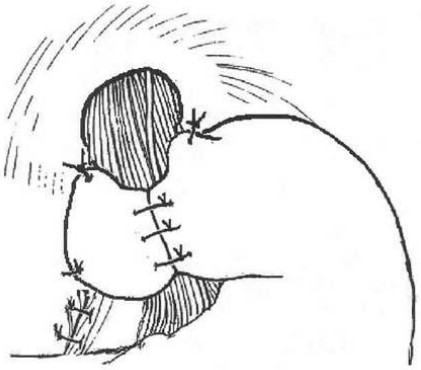
Another multicenter RCT done by Grant et al comparing surgical treatment with medical treatment in patients documented with GERD, involved individuals who were on medical management for a median of nearly 32 months. This study reported that at the end of 1 year, 38% of

those post surgery were taking reflux medication compared to 90% of the individuals randomized to medical management. Also, long-term results of laparoscopic Fundoplication have revealed that at end of 10 years, 90% of patients were symptom free with only a minority still having to take PPIs.[27]

Another Long-term followup from a multicenter, pragmatic randomized trial revealed that relative to pharmacotherapy, post surgical patients maintained better symptomatic relief in gastro oesophageal reflux without evidence of any adverse long-term postsurgical symptoms. Results of this study concluded that at the end of 5 years, nearly twice as many patients randomized to medical management (82%) were taking antireflux agents compared to those who had been randomized to surgery (44%). [25,26]

Laparoscopic fundoplication (Nissens floppy 360 degree fundoplication)

Laparoscopic Nissens fundoplication is generally performed under GA. Five 5 to 10mm port incisions are made. The basic surgical principle is the fundus of the stomach being wrapped around the lower oesophagus creating a new valve at the level of the oesophagogastric junction.



The important surgical principles are as follows:

- All around completely mobilise the fundus of stomach with or without division of the short gastric vessels
- Repair of hiatus hernia
- Narrowing the oesophageal hiatus
- Creating a floppy 360° fundoplication over a Oesophageal bougie (dilator)

Laparoscopic fundoplication generally lasts 2 - 2 1/2 hours. The mean postoperative hospital stay is roughly 2 days. Patients usually resume normal activities in 2-3 weeks. According to study, approximately 92% of patients are fully resolved of their symptoms post surgery. [18]

PPIs, though useful to control the acid component of reflux, does not completely rid the patient of bile reflux, which is a major contributor of Barrett's epithelium. Subjects with Barrett's oesophagus usually have lower LES pressures and comparatively poorer oesophageal peristalsis

than in those without Barrett's. Hence they are also subjected to a large quantity of reflux. Here Nissens floppy fundoplication offers the only possibility to reduce reflux by recreating a competent LES.

Partial fundoplication :

- 270 posterior – **Toupet**

In modified toupet the fundus is mobilized posterior to oesophagus as in Nissens fundoplication. The posterior wall of the fundus on each side of the oesophagus is sutured to oesophagus with interrupted 2'0 or 3'0 absorbable sutures approximately 45 degress to either side of the mid anterior oesophageal wall. A total of 3 interrupted sutures are placed on each side approximately 1cm apart resulting in a 2 cm fundoplication. Care should be taken to position the fundoplication on the oesophagus and not onto the stomach and to preserve the vagus, not entrapping it within the suture

- 180 anterior – **Dor** and

Here the fundoplication is constructed by accentuating the angle of His and folding the fundus over the anterior surface of oesophagus. The first suture fixes the medial wall of the fundus near the Angle of His to the anterior aspect of the left crus. The next suture is placed between the fundus and left anterior aspect of the hiatus. The fundus is then folded

over the anterior aspect of the hiatus by placing a suture between the fundus and right anterior aspect of hiatus. Several additional sutures are placed approximately at 1cm intervals, approximating the fundus of stomach to the right posterolateral wall of the oesophagus and right crus

- 270 gastric fundoplication perfomed through chest – **Belsey Mark 4**

In this procedure which is performed transthoracic the oesophagus is adequately mobilized to avoid tension. In situations where the oesophagus has shortened, aggressive mobilization does not produce a tension free repair. To counter this, oesophageal lengthening technique was done by making a tube about the diameter of the oesophagus and 5cm in length in the lesser curvature of the stomach and constructing a Belsey fundoplication around the tube. This was called the Collin Belsey repair named after Dr. Leigh Collin and Dr. Ronald Belsey. The major drawback of this procedure is acid production within the tubularised portion of the stomach. Also this procedure is difficult to teach and has very less margin for error. In an experienced surgeon Mark Belsey operation has similar success to Nissens fundoplication.

Complications of antireflux surgery :

- Most common perioperative complication is early wrap herniation (1.3%) occurring within 48 hours of surgery. It is more common in

Laparoscopic than open surgeries, reason being attributable to opening of more tissue planes due to pneumoperitoneum and reduced tendency for adhesion formation in laparoscopic surgery. It is avoided by performing crural repair.

- Pneumothorax & Pneumperitoneum : Pneumothorax is attributable to breach of either of the pleural membrane, more commonly the left during hiatal dissection. Chest drain insertion is rarely needed as Co2 rapidly dissipates following the release of pneumoperitoneum due to absorption and positive pressure ventilation.
- As with any laparoscopic procedures hollow viscous perforation may occur. Oesophageal perforation may occur during passage of Bougie, retrooesophageal dissection or suture pull through. Late oesophageal perforation may be related to Diathermy use during mobilization. Gastric perforations usually occur due to excessive traction on the fundus for retraction purposes.
- Hemorrhage during surgery arises mostly from Short gastric vessels or spleen. Retractor trauma to liver, Injury to left inferior phrenic vein, Aberrant Left hepatic artery and Inferior vena cava are rare causes. Similarly cardiac tamponade due to ventricular trauma has also been reported.

- Slipped or Misplaced Nissen - A "slipped Nissen" occurs when the body of stomach intussuscepts through the fundoplication, creating an hourglass deformity where both above and below the wrap stomach is residing. This complication is associated with severe regurgitation and reflux, since the pouch of stomach residing above fundoplication can trap food and serves as a reservoir for acid-rich refluxate below an incompetent lower esophageal sphincter (LES). Also, the wrap can get misplaced around upper stomach rather than oesophagus, creating an hourglass defect where the wrap is below the diaphragmatic hiatus and the upper stomach and gastrooesophageal junction above the diaphragmatic hiatus.
- Twisted Fundoplication – This occurs when the short gastric vessels are not divided, although it can also happen when they are divided. Here a portion of anterior stomach wall is pulled from the left around the oesophagus posteriorly and sutured to another portion of the anterior stomach wall that has been pulled from a spot low on the greater curvature. This can create tension at the GEJ which causes rotation of the distal oesophagus with the fundoplication causing a spiral-type deformity seen in retroflexion of an endoscope. This deformity usually can produce dysphagia and severe postoperative gas bloat that do not respond to dilatation.

The fundoplication valve is twisted by the fundus attempting to unwind itself

- Post operative gastrooesophageal Reflux disease : Post operative gastro oesophageal reflux and dysphagia are related to the length of myotomy and type of antireflux procedure and not to the approach for the procedure (open or laparoscopic) It is usually mild and can be managed medically
- Post operative dysphagia
- Weight loss
- Gas bloat

One complication which has been virtually eliminated post the Laparosopic era is splenic injury

"Medical versus surgical management for gastro-oesophageal reflux disease in adults" done by Wileman SM, McCann S, Grant AM, Krukowski ZH, Bruce J. , University of Aberdeen, Foresterhill, Aberdeen, UK by comparing the effects of medical management versus laparoscopic fundoplication surgery using health-related and GORD-specific quality of life (QOL) in adults with GORD, concluded a Significant decrease in antacid medication use **(97% vs 19%)** and that laparoscopic fundoplication surgery is more effective than medical

management for the treatment of GORD atleast in the short to medium term. Surgery does carry some risk but the benefits outweigh the risks [12]

"Quality of life following laparoscopic Nissen fundoplication: Assessing short-term and long-term outcomes" done by Ilmo Kellokumpu, Markku Voutilainen, Caj Haglund, Martti Färkkilä, Peter J Roberts, Hannu Kautiainen, published in world journal of Gastroenterology, Jun 2013 done for two hundred and forty-seven patients for short-term analysis following endoscopy showed **cure** of Gastroesophageal reflux disease **in 98.4%** of patients three months after surgery. **New-onset dysphagia** was encountered postoperatively in 13 patients **(6.7%)**; **95%** reported that the outcome was better after antireflux surgery than with preoperative medical treatment. [28]

"Comparative Effectiveness of Management Strategies For Gastroesophageal Reflux Disease" was done by Stanley Ip, MD, Joseph Lau, MD in 2005 Dec, Tufts-New England Medical Center EPC, Boston, Massachusetts. They did a 10-year study of open fundoplication (Spechler) included mostly patients with complicated GERD who received non-PPI based medical interventions at enrollment, a 5-year study of open fundoplication (Lundell) included patients whose symptoms and oesophagitis responded to PPI treatment, and a 1-year study of laparoscopic fundoplication (Mahon) included mostly patients

with GERD symptoms but without complications who had been treated with PPIs for at least 3 months. The two studies of open fundoplication (Spechler and Lundell) reported similar degree of improvement in symptoms compared with baseline in the medical group and significant reduction in oesophageal acid exposure in the surgical group compared to medical therapy at three months in the study by Mahon and one year in the study by Spechler. [2]

"Surgical Management of Extra-esophageal Reflux Disease" done during the Grand Rounds Presentation in the Department of Otolaryngology in The University of Texas Medical on January 29, 2013 by Andrew Coughlin, MD,. Michael Underbrink, MD and Francis B. Quinn, Jr., MD say that a conservative approach with empiric therapy can be helpful prior to ordering expensive tests. If patients continue to be symptomatic then a formal workup with EGD to rule reflux is instituted. Other tests like impedance and manometry should be reserved for patients with more severe oesophageal problems. Referral to surgeons for Nissen's fundoplication should be considered in all patients with documented reflux who are resistant to therapy, have proven complications or do not wish to take medications for life. [31]

"Symptoms and Antireflux Medication Use Following Laparoscopic Nissen Fundoplication outcome at 1 and 4 Years" published by Mark Bloomston, MD, William Nields, BA, Alexander S. Rosemurgy, MD published in **JSLS**, In this study one hundred patients undergoing laparoscopic Nissen fundoplication between 1992 and 1997 were asked, at 1 to 2 years and 4 to 6 years postoperatively, to grade their symptoms on a scale of 1 (mild) to 10(severe). Patients were also queried as to the number/cost of antacid medications used before and after fundoplication. Laparoscopic Nissen fundoplication results in a significant reduction in the symptoms of reflux and the use of antacid medications with a high degree of patient satisfaction. Although some patients return to antacid medications at late follow-up, they continue to have few symptoms and are pleased with their outcomes. [27]

"Laparoscopic Nissen fundoplication - 200 consecutive cases", done by D C Gotley, B M Smithers, M Rhodes, B Menzies, F J Branicki, L Nathanson published in GUT journal. 200 patients operated between 1991 and 1994 were included in the study. Pre-operative assessment included symptom score, endoscopy, manometry and 24 hour pH monitoring of the oesophagus. Patients were evaluated at three and 12 months after surgery. They came to the conclusion Laparoscopic Nissen fundoplication is a safe and effective procedure for gastro-oesophageal

reflux disease. With experience, the duration of operation falls and the hospital stay is shorter. Short term symptomatic and pH results show a consistent improvement by surgery. [36]

According to a scientific paper published in JSLS – **"Symptoms and Antireflux Medication Use Following Laparoscopic Nissen Fundoplication: Outcome at 1 and 4 Years"** by Mark Bloomston, MD, William Nields, BA, Alexander S. Rosemurgy, MD, Laparoscopic Nissen fundoplication results in Significant decrease in antacid medication use **(97.3% vs 18.4%)** in a significant reduction in the symptoms of reflux and the use of antacid medications with a high degree of patient satisfaction. Although some patients return to antacid medications at late follow-up, they continue to have few symptoms and are pleased with their outcomes.[34]

Another **"Prospective Study of Laparoscopic Nissen Fundoplication in a Community Hospital and Its Effect on Typical, Atypical, and Nonspecific Gastrointestinal Symptoms"**published in JSLS done by Mark E. Ranson, MD, Amanda Danielson, MS, J. Gary Maxwell, MD, James A. Harris, MD studied 91 patients and long-term data on 84 patients were studied. Overall long-term improvement was **98%**. Regarding typical symptoms, the greatest improvement occurred in heartburn and regurgitation. Regarding atypical symptoms, the greatest

improvement occurred in cough and sore throat. In this study Bloating, nausea, and diarrhea showed no significant change between preoperative and postoperative surveys. Mild weight loss was common.

According to another scientific paper published in JSLS, **"Postoperative Gastrointestinal Complaints After Laparoscopic Nissen Fundoplication"** done by Constantine T. Frantzides, MD, PhD, Mark A. Carlson, MD, John G. Zografakis, MD, Ronald E. Moore, MD, Tallal Zeni, MD, Atul K. Madan, MD patients who have undergone laparoscopic Nissen fundoplication for gastrooesophageal reflux disease will have gastrointestinal complaints mostly during the initial 3 postoperative months. Nearly all of these patients, aproximately **95%** have resolved symptomatology after 3 months. Those with persistent symptoms after 3 months warrant evaluation for operative failure. [32]

MATERIALS AND METHODS

MATERIALS

A Prospective / Retrospective study conducted in PSG Institute of Medical Sciences and Research to assess whether Laparoscopic fundoplication really improves the quality of life compared to medical management and to assess the usefulness of surgery based on risk vs benefit ratio by comparison of their preoperative and postoperative Quality of life with a questionnaire and endoscopic findings in 31 patients

QUESTIONNAIRE

NAME:

AGE:

SEX:

ADDRESS:

IP NO/ OP NO:

DOA:

DOS:

DOD:

PRE OPERATIVE:

HISTORY (ONSET, DURATION AND COMPLAINTS) :

PAST / PERSONAL H/O:

MEDICATIONS HE/SHE IS TAKING FOR THE COMPLAINTS

PHYSICAL EXAMINATION

UGI SCOPY REPORT:

USG ABDOMEN (IF DONE) :

OESOPHAGEAL MANOMETRY(IF DONE)

COMPLAINTS Graded on **GERD - HRQL (HEALTH RELATED QUALITY OF LIFE SCALE)**

- **HEARTBURN** (each 1 point)

On lying down

On standing position

After meals

Does it disturb sleep

- **DIFFICULTY IN SWALLOWING**
- **PAIN ON SWALLOWING**
- **BLOATY FEELING**
- **ABDOMINAL PAIN**

(2nd-5th) 1-5 (TICK WHATEVER IS APPLICABLE) points

0 - NO SYMPTOMS

1 - SYMPTOMS PRESENT, NOT BOTHERSOME

2 - SYMPTOMS PRESENT, NOT EVERYDAY BOTHERSOME

3 - SYMPTOMS PRESENT, EVERYDAY BOTHERSOME

4 - SYMPTOMS AFFECTING DAILY ACTIVITIES

5 - SYMPTOMS INCAPACITATING

Scoring (MAXIMUM OF 29, MINIMUM 0) – PROGNOSIS BETTER, THE LESSER THE SCORE

ANTIREFLUX MEDICATION (1 point for each)

- H2 blockers
- Antacids
- Ca channel blockers
- Anti anxiety drugs
- Prokinetics

Type of Study

Observational, Prospective and Retrospective

Inclusion Criteria

Clinical reflux symptoms

UGI endoscopy

Exclusion Criteria

H/o previous oesophageal surgery

Complicated reflux (eg. Ulcer, Short oesophagus)

METHODOLOGY

In this study 30 patients who had clinical symptoms of gastro oesophageal reflux were included.

Informed consent was obtained detailing the study process, need for the study and its benefits, elaborately. The history was recorded by the principal investigator.

The questionnaire was filled diligently by the principal investigator.

All 31 patients were subjected to clinical examination by the principal investigator in an unbiased manner.

Their Upper GI endoscopy findings were recorded, along with Oesophageal manometry and USG abdomen as and when required. Clinical symptoms of patients, especially those refractory to long term medical management and findings with UGI scopy played a role in patient selection for surgery.

After the patients underwent laparoscopic fundoplication, re evaluation of their symptoms and their quality of life was done with History, clinical examination and questionnaire and repeat UGI scopy.

RESULTS AND OBSERVATION

RESULTS

		Pre operative									Post operative								
		1A	1B	1C	1D	2	3	4	5	MED	1A	1B	1C	1D	2	3	4	5	MED
1.	I12026998	1	0	1	1	3	3	1	2	2	Not	pr	es	en	te	r	for	fol	low up
2.	I12027342	1	0	0	0	1	0	3	1	2	0	0	0	0	0	0	0	0	0
3.	I12027457	1	0	1	0	2	2	0	0	1	0	0	0	0	1	0	0	0	1
4.	I12031732	1	0	1	0	2	2	3	0	2	0	0	0	0	0	0	0	0	0
5.	I12080549	1	0	1	1	3	2	0	2	1	0	0	0	0	0	0	0	0	0
6.	I12035901	1	0	1	1	2	2	4	4	2	0	0	0	0	0	0	2	0	2
7.	I12036244	1	0	1	1	0	2	2	4	1	0	0	0	0	0	0	0	0	0
8.	I13001068	0	0	1	0	1	1	2	3	1	0	0	0	0	0	0	0	1	1
9.	I13001741	0	0	1	0	2	0	2	4	1	0	0	0	0	0	0	0	1	1
10.	I13002631	1	0	1	0	2	0	3	4	2	0	0	0	0	0	0	0	0	0
11.	I13010639	0	0	0	0	0	0	3	4	1	0	0	0	0	0	0	0	0	0
12.	I13015655	1	1	0	1	3	1	4	1	2	0	0	0	0	0	0	2	0	2

		Pre operative									Post operative								
		1A	1B	1C	1D	2	3	4	5	MED	1A	1B	1C	1D	2	3	4	5	MED
13.	I13018222	1	1	1	1	2	2	0	3	2	0	0	0	0	0	0	0	0	1
14.	I13021791	1	1	1	1	1	0	1	4	2	0	0	1	0	0	0	0	0	0
15.	I13022366	0	1	1	1	2	1	4	3	1	0	0	0	0	0	0	0	0	0
16.	I13025914	1	1	1	1	2	2	2	4	1	0	0	0	0	0	0	0	0	0
17.	I13026979	1	0	1	0	2	1	2	4	1	0	0	0	0	0	0	0	0	0
18.	I13028923	1	0	1	0	2	2	4	4	1	0	0	0	0	0	0	0	0	0
19.	I13029681	0	0	1	1	0	2	4	4	2	0	0	0	1	0	0	0	2	1
20.	I13030107	1	0	1	1	4	2	0	0	2	0	0	0	0	3	0	0	0	2
21.	I13034472	0	1	1	1	3	3	2	3	1	0	0	0	0	1	0	0	0	1
22.	I13036039	1	1	0	1	3	3	4	3	3	0	0	0	0	0	2	0	0	1
23.	I13035829	0	0	1	0	0	2	2	4	1	0	0	0	0	1	0	0	0	0

		Pre operative									Post operative								
		1A	1B	1C	1D	2	3	4	5	MED	1A	1B	1C	1D	2	3	4	5	MED
24.	I13038462	1	1	1	1	3	3	3	4	2	0	0	0	0	0	1	1	0	0
25.	I14000025	0	0	1	0	0	0	2	4	1	0	0	0	0	0	0	0	0	0
26.	I14002644	0	1	1	1	3	5	3	4	1	0	0	0	0	0	0	0	0	0
27.	I14005842	1	1	0	1	2	2	2	4	2	0	0	0	0	0	0	2	0	1
28.	I14022873	1	1	1	1	1	3	1	3	1	0	0	0	0	0	0	1	0	0
29.	I14024343	1	1	1	1	2	2	4	4	2	0	0	0	0	0	0	1	0	1
30.	I15006931	0	0	1	1	3	3	2	3	2	0	0	0	0	0	0	0	0	1
31.	I15022648	1	0	1	1	2	2	0	0	1	0	0	0	0	1	0	0	0	0

		Pre operative				Post operative			
		UGI Scopy	24 hr pH	Oesop -hageal Manom -etry	USG	UGI Scopy	24 hr pH	Oesop -hageal Manom -etry	USG
1.	I12026998	Hiatal Hernia, Lax LES, Mucosalprolaspe	-	-	N Study	NOT PRESENTED FOR FOLLOW UP			
2.	I12027342	Hiatus hernia, Gastric Mucosal prolapse, Gr 2 Reflux oesophagitis	-	-	N study	Post fundo wrap intact			
3.	I12027457	Hiatus hernia e Gr 2 Reflux Oesophagitis	-	Low sphincter pressure. 100% motility	N Study	Wrap intact, Gr 1 oesophagitis, Antral gastritis			
4.	I12031732	Grade 3 reflux oesophagitis, Antral gastritis	-	-	N Study	Post fundo wrap intact			
5.	I12080549	Hiatus hernia, Gr 4 reflux oesohagitis	-	-	N study	Post fundo wrap intact			
6.	I12035901	Hiatus hernia	-	-	N study	Post fundo wrap intact			
7.	I12036244	Hiatus hernia	-	-	N study	Post fundo wrap intact			
8.	I13001068	Hiatus hernia, Antral gastritis, Duodenitis	-	-	N study	Wrap intact, Gr 1 oesophagitis, Antral gastritis			

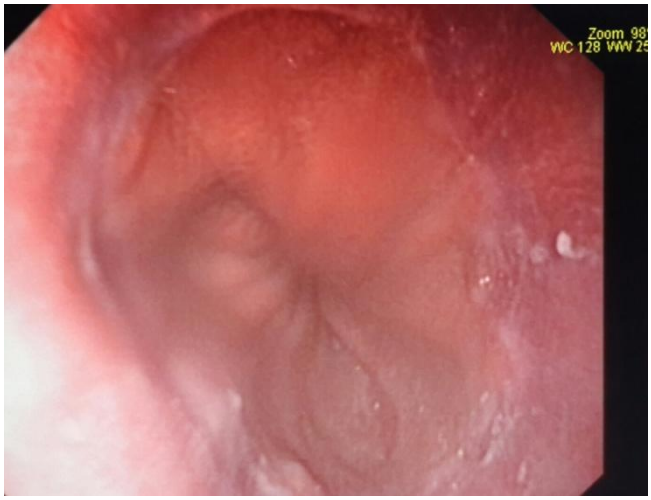
		Pre operative				Post operative			
		UGI Scopy	24 hr pH	Oesop -hageal Manom -etry	USG	UGI Scopy	24 hr pH	Oesop -hageal Manom -etry	USG
9.	I13001741	Hiatal Hernia, Duodenal ulcer	-	-	N Study	Wrap intact, Gr 1 oesophagitis			
10.	I13002631	Hiatus hernia, Grade 2 Reflux oesophagitis	-	-	N study	Post fundo wrap intact			
11.	I13010639	Hiatus hernia e Gastric mucosal prolapse	-	-	N Study	Post fundo wrap intact			
12.	I13015655	Hiatus hernia	-	-	N Study	Wrap intact, Gr 1 oesophagitis			
13.	I13018222	Hiatus hernia, Gastric mucosal prolapse	-	-	N study	Post fundo wrap intact			
14.	I13021791	Lax hiatus, Antral gastritis	-	-	N study	Post fundo wrap intact			
15.	I13022366	Hiatus hernia	-	-	N study	NOT WILLING FOR FOLLOWUP ENDOSCOPY			
16.	I13025914	Hiatus hernia	-	-	Minimal ascites	Post fundo wrap intact			
17.	I13026979	Hiatus hernia, Gr 3 Reflux oesophagitis	-	-	N study	Post fundo wrap intact			

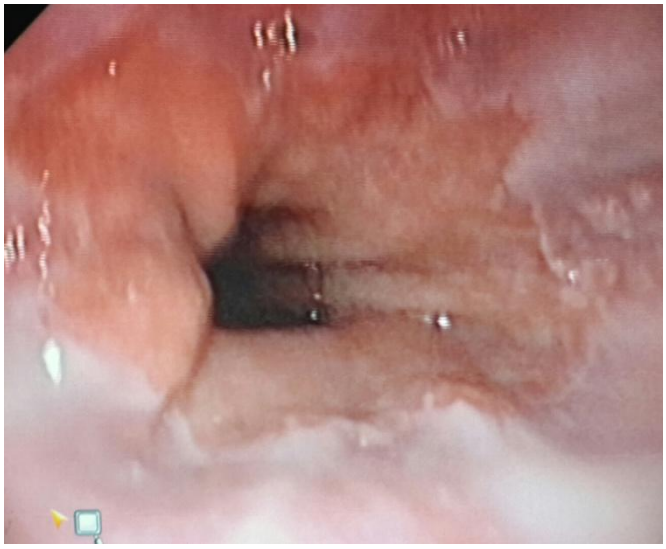
		Pre operative				Post operative			
		UGI Scopy	24 hr pH	Oesop -hageal Manom -etry	USG	UGI Scopy	24 hr pH	Oesop -hageal Manom -etry	USG
18.	I13028923	Hiatal Hernia, Gr 1 Reflux oesophagitis, Antral gastritis	-	-	N Study	Post fundo wrap intact			
19.	I13029681	Hiatus hernia, Antral gastritis, Duodenitis	-	-	N study	Prolapse gastropathy, erosive gastroduodentits Wrap intact			
20.	I13030107	Hiatus hernia type 3	-	-	N Study	Gr A Reflux oesophagitis, Mild scalloped duodenal margins, No hiatus hernia			
21.	I13034472	Hiatus hernia	-	Sphincter pressure N. 100% motility	Rt renal calculus	Wrap intact, Gr 1 oesophagitis			
22.	I13036039	Lax hiatus	-	-	Minimal ascites	Wrap intact, Gr 1 oesophagitis			
23.	I13035829	Lax LES, Sliding Hiatus hernia, Gr 1 oesophagitis	-	-	N study	Post fundo wrap healthy			
24	I13038462	Hiatus hernia	-	-	N study	Gr 1 oesophagitis, wrap intact			

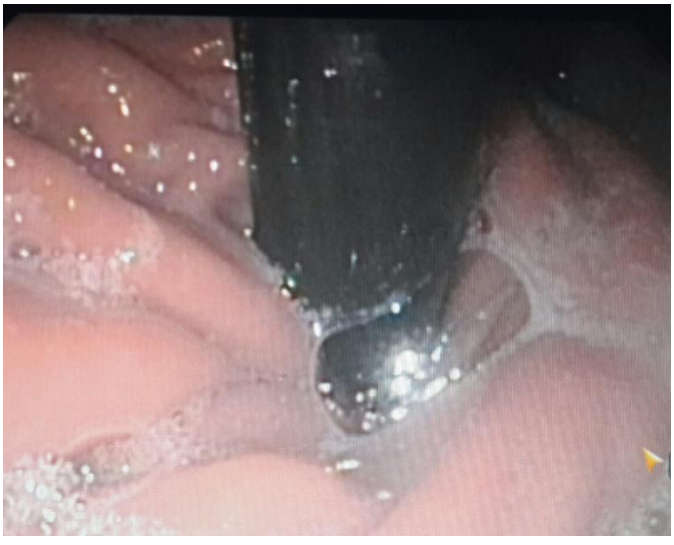
		Pre operative				Post operative			
		UGI Scopy	24 hr pH	Oesop -hageal Manom -etry	USG	UGI Scopy	24 hr pH	Oesop -hageal Manom -etry	USG
25.	I14000025	Gr 1 oesophagitis Lax LES, Erosive gastritis, Extrinsic impression of fundus on body of stomach	-	Hypotensive LES	N Study	NOT WILLING FOR FOLLOWUP ENDOSCOPY			
26.	I14002644	Hiatus hernia	-	-	N study	Post fundoplication wrap intact,			
27.	I14005842	Hiatus hernia Antral gastritis, prepyloric ulcer	-	-	B/l mild pleural effusion Rt simple ovarian cyst	Gr 1 oesophagitis. Fundo wrap intact, antral gastritis,			
28.	I14022873	Hiatus hernia	-	-	Minimal perihepatic fluid	Gr 1 reflux oesophagitis Post fundoplication status partial wrap, small hiatus hernia			
29.	I14024343	Hiatus hernia, prolapse gastropathy, Antral gastritis	-	Hypotensive LES	N study	Prolapse gastropathy, post fundo wrap healthy			

		Pre operative				Post operative			
		UGI Scopy	24 hr pH	Oesop -hageal Manom -etry	USG	UGI Scopy	24 hr pH	Oesop -hageal Manom -etry	USG
30.	I15006931	Hiatus hernia Lax LES, Gr 3 reflux oesophagitis, prolapse gastropathy	-	Hypotensive LES	N Study	Fundo wrap intact, antral gastritis, Gr 1 reflux oesophagitis			
31.	I15022648	Hiatus hernia Antral gastritis,	-	-	N study	Post fundoplication - wrap intact			

Pre and Post operative endoscopy pictures taken in our hospital







OBSERVATION

Analysis of Data:

A total of 31 cases were included in the study

1 patient did not present for follow up

For 30 cases informed written consent was obtained

For all 30 cases preoperative and post operative history was elicited and questionnaire completed

For 31 cases preoperative UGI scopy done

For 5 cases preoperative Oesophageal manometry was done

For 28 patients post operative USG was done. (2 patients were not willing for follow up USG)

Pre operatively all 31 patients were taking either PPI's, Antacids, Prokinetics or the combination of above. Post operatively 16 patients were still taking PPI's. (It must be noted that there was marked relief of symptoms, with medication post Surgery compared to prior to surgery, in which a majority of patients were refractory to medical management). This compares favourably with the international standard of patients on pre and post surgical medication)

According to the Health Related Quality of Life questionnaire, the relief of symptoms post surgical management is 93% which again compares favourably with international standards

According to UGI scopy findings

10 subjects had Grade 1 reflux oesophagitis post surgery

3 patients among them had antral gastritis

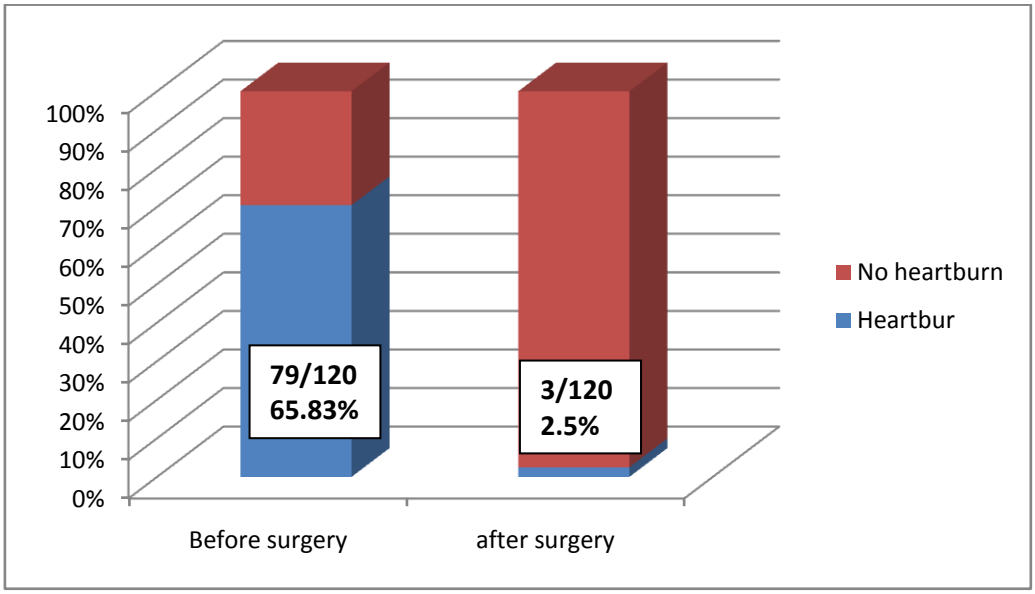
1 patient had erosive gastritis

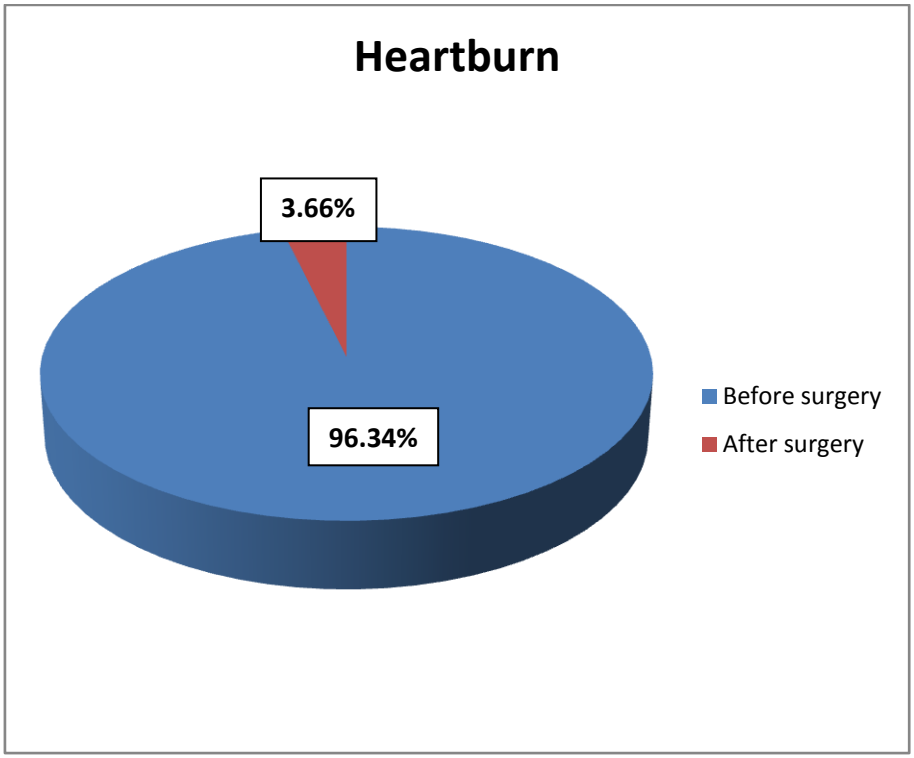
3 patients had prolapse gastropathy

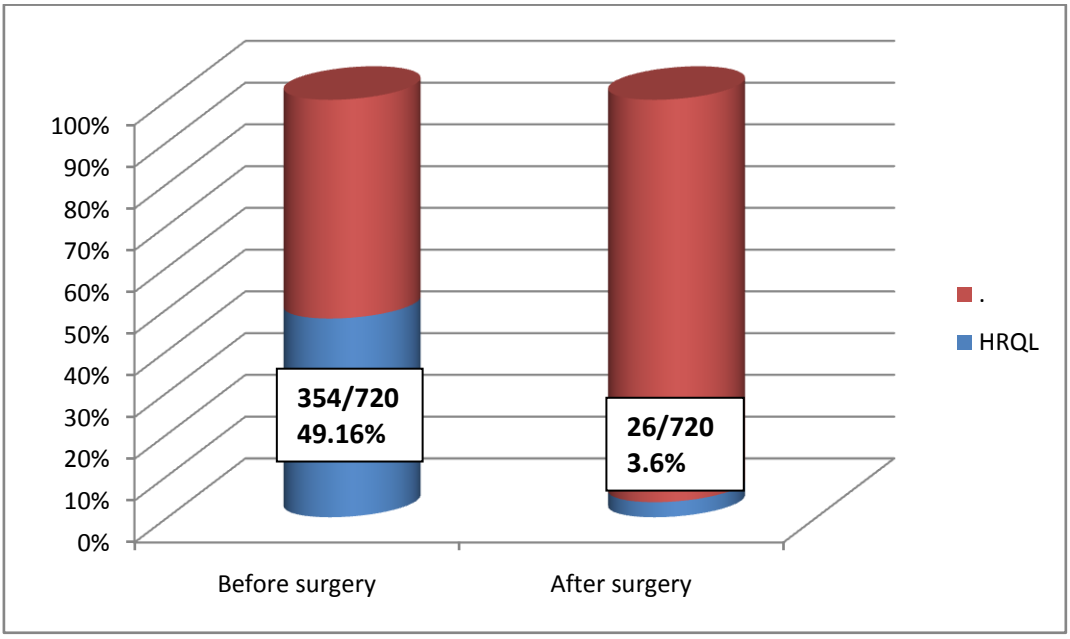
1 patient had a small hiatal hernia post surgery

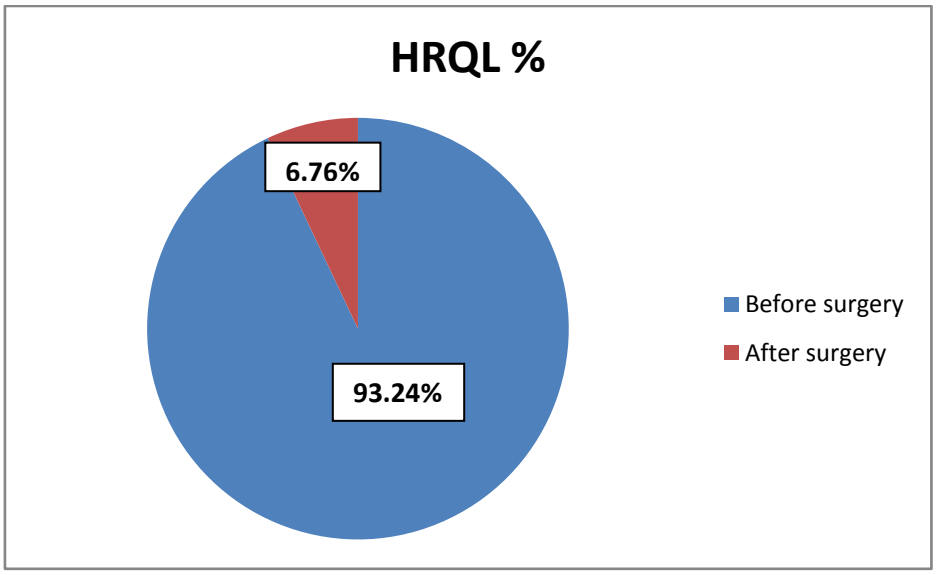
Rest of 27 patients had intact fundoplication wrap

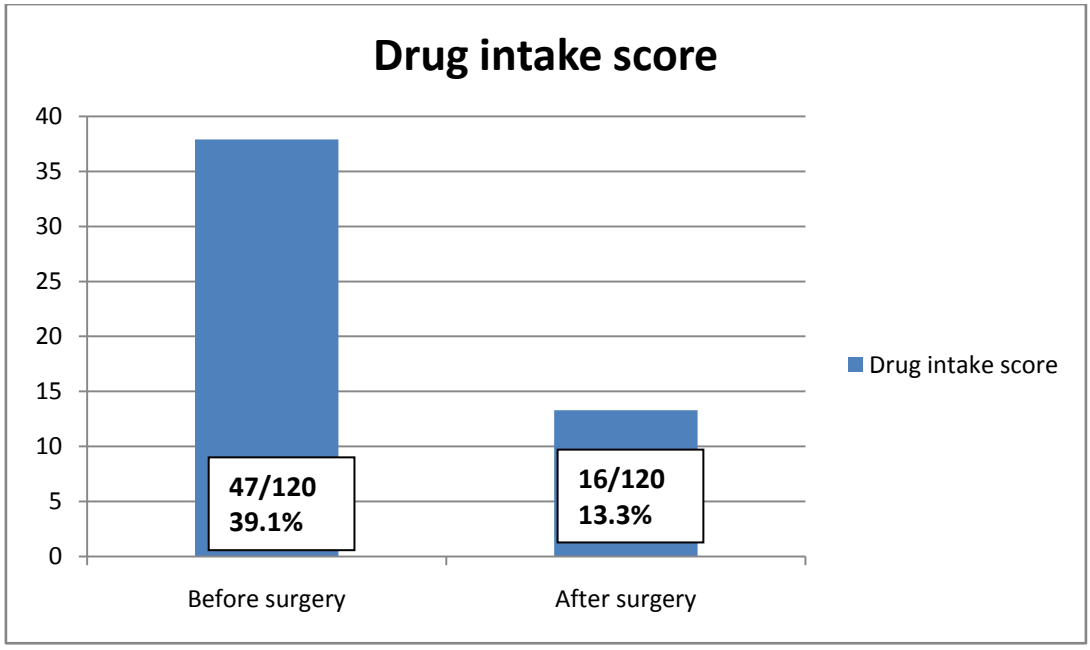
These recurrence rates are on par or better in comparison with international standards

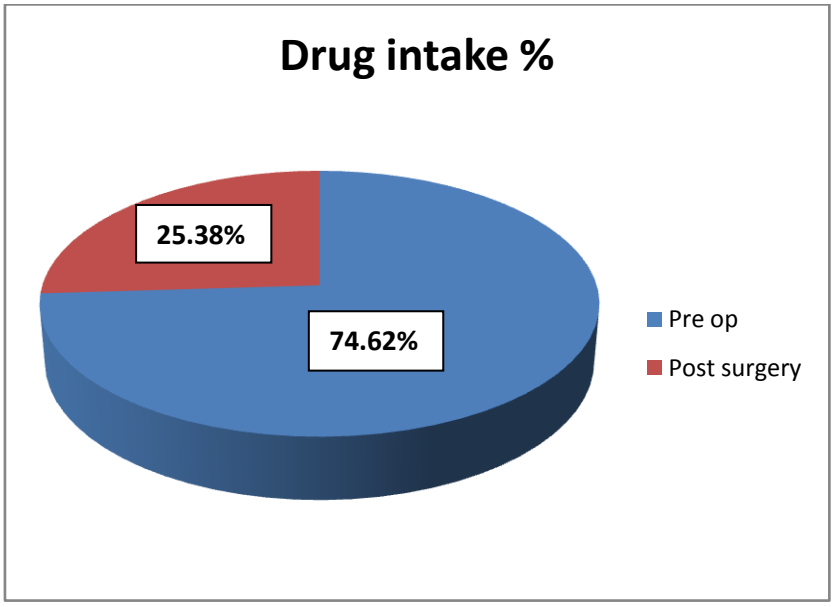

100%
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
79/120
65.83%
3/120
2.5%
Before surgery
after surgery
No heartburn
Heartbur

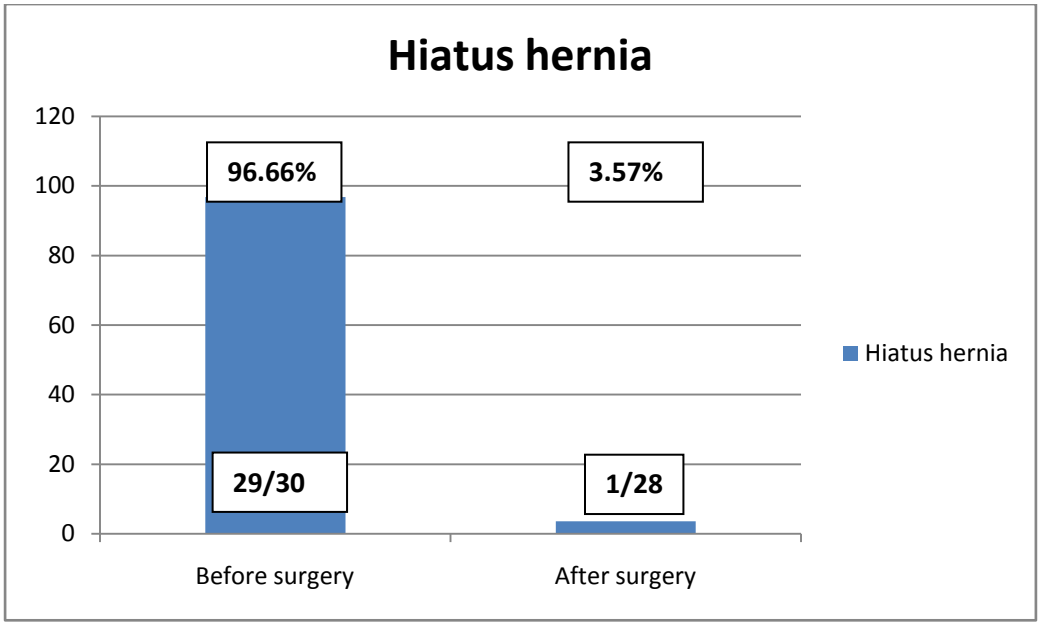

Heartburn
3.66%
96.34%
Before surgery
After surgery

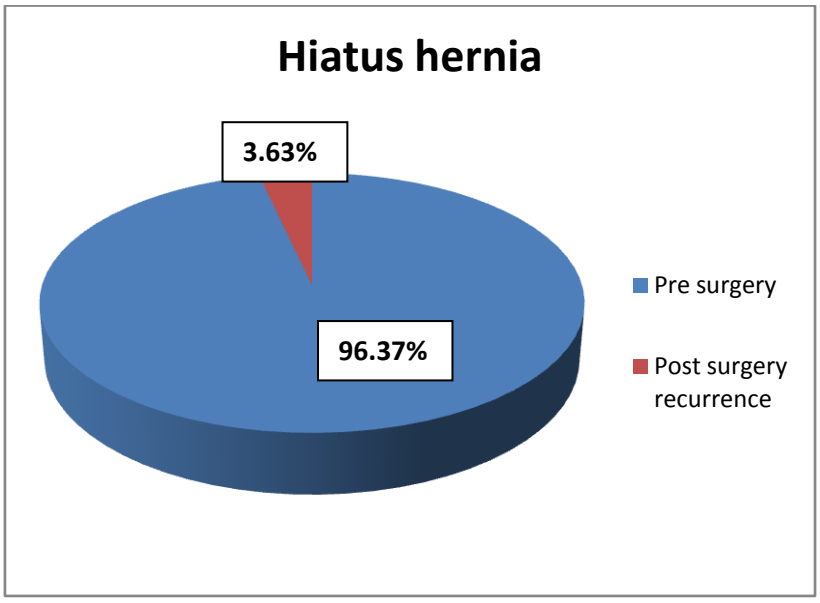

100%
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
354/720
49.16%
26/720
3.6%
Before surgery
After surgery
.
HRQL


HRQL %
6.76%
93.24%
Before surgery
After surgery


Drug intake score
40
35
30
25
20
15
10
5
0
47/120
39.1%
16/120
13.3%
Before surgery
After surgery
Drug intake score


Drug intake %
25.38%
74.62%
Pre op
Post surgery


Hiatus hernia
120
100
80
60
40
20
0
96.66%
3.57%
29/30
1/28
Before surgery
After surgery
Hiatus hernia


Hiatus hernia
3.63%
96.37%
Pre surgery
Post surgery recurrence

DISCUSSION

DISCUSSION

A prospective / retrospective study of evaluation of 30 patients undergoing laparoscopic nissens fundoplication in PSG hospitals was done and it infers the following.

This study expands from previous research done in evaluating the patients who had undergone laparoscopic Nissens fundoplication and we have concluded our experience with our study population which exhibits particular similarities and differences compared to that of international research data available

1. Post operative improvement and relief of heartburn ranged from complete relief to 90% (at the end of a ten year study). This compares favourably with our data where our patients give **96.3%** improvement in the relief of heart burn post fundolication [13,45,46,47,55]

2. HRQL (quality of life score) improvement as per our study was **93.2%**. This again compares favourably with international standards which ranges between 91% to 95% according to various studies. [14,16,23,24,28,32, 55]

3. **10%** of our patients had post operative dysphagia. This is lesser than international literature which reports between 25-39%.[16,31,32,52] Proper surgical technique plays a role in this.
4. Recurrence of Hiatal hernia as per our study was **3.7%**. This was again lesser than international standards which was between 10-12%. Proper surgical technique plays a role in preventing recurrence. It is also noted that our study population and period was relatively smaller than international literature [34,47]
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Inspite of our study numbers being small and of shorter duration, we can conclude that our overall experience in evaluation of the patients who had undergone laparoscopic Nissen's fundoplication, showed a significant reduction and relief of reflux symptoms. The proportion of patients who had to rely on medication post surgery was also significantly less than prior to surgery. Even if the patients relying on medication were slightly higher than international standards it is worth noting that post surgery these patients have relief through PPI's compared to those prior to surgery who were refractory to medical management.

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APPENDIX

PROFORMA

QUESTIONNAIRE :

NAME:

AGE:

SEX:

ADDRESS:

IP NO/ OP NO:

DOA:

DOS:

DOD:

PRE OPERATIVE:

HISTORY (ONSET, DURATION AND COMPLAINTS) :

PAST / PERSONAL H/O:

MEDICATIONS HE/SHE IS TAKING FOR THE COMPLAINTS

PHYSICAL EXAMINATION

UGI SCOPY REPORT:

USG ABDOMEN (IF DONE) :

OESOPHAGEAL MANOMETRY(IF DONE)

COMPLAINTS Graded on **GERD - HRQL (HEALTH RELATED QUALITY OF LIFE SCALE)**

- **HEARTBURN** (each 1 point)

  On lying down

  On standing position

  After meals

  Does it disturb sleep

- **DIFFICULTY IN SWALLOWING**
- **PAIN ON SWALLOWING**
- **BLOATY FEELING**
- **ABDOMINAL PAIN**

(2nd-5th) 1-5 (TICK WHATEVER IS APPLICABLE) points

0 - NO SYMPTOMS
1 - SYMPTOMS PRESENT, NOT BOTHERSOME
2 - SYMPTOMS PRESENT, NOT EVERYDAY BOTHERSOME
3 - SYMPTOMS PRESENT, EVERYDAY BOTHERSOME
4 - SYMPTOMS AFFECTING DAILY ACTIVITIES
5 - SYMPTOMS INCAPACITATING

Scoring (MAXIMUM OF 29, MINIMUM 0) – PROGNOSIS BETTER, THE LESSER THE SCORE

ANTIREFLUX MEDICATION (1 point for each)

- H2 blockers
- Antacids
- Ca channel blockers
- Anti anxiety drugs
- Prokinetics

ABSTRACT

AN EVAULATION OF PATIENTS UNDERGOING LAPAROSCOPIC NISSENS FUNDOPLICATION

This dissertation is submitted to PSG Institute of Medical Sciences and Research in partial fulfillment of the regulations for the M.S (General Surgery) Degree Examination, April 2016

By

Dr. PRASANNA KUMAR S

Done under the guidance of

Dr.Balashanmugam.T.S

Professor of Surgery

PSG IMS&R

Coimbatore – 641004

INTRODUCTION

Over years the effective treatment for GERD has been antireflux medication and surgery. Patients who were not improving on medical therapy or are not conducive are nowadays surgically treated. This technique was originally pioneered by Rudolf Nissen. In a young male patient with penetrating oesophageal ulcer he did the first fundoplication to protect the oesophagogastric anastamosis . As he followed up the patient he noticed that the patient's reflux symptoms were no more present. Nissen again reattempted his fundoplication on a male patient with a paraesophageal hernia which had incarcerated. It produced excellent clinical results. Eventually he published the first description of his surgical procedure in 1956, which turned out to be the birthchild of the current era of antireflux surgery.

In original Nissen's fundoplication, he united the posterior wall of the stomach with the anterior wall completely around the fundus of the stomach to provide a full 360-degree wrap of 4-5cm around the lower esophagus. One or two stitches should include the wall of the esophagus to prevent slippage of the cardia. In Nissen-Rossetti's modification of the original fundoplication, he used only the anterior wall of the fundus and constructed a 360-degree wrap enclosing the distal oesophagus. In the initial Nissen-Rossetti modification he did not divide the short gastric vessels. However, when a tension-free wrap could not be obtained, the short

gastric vessels were sacrificed. The role of Operative treatment for Gastro Oesophageal Reflux Diseases and Hiatus Hernias have changed dramatically during the 90's. The driving force behind increased surgical numbers is the development of minimal invasive surgery. [3,4,5]

Although the techniques of Antireflux surgery has not changed, the approach has become more acceptable to the patient and referring physician because of small incisions, relatively short hospital stay and lack of post operative pain when compared to open approach.

Aims and Objective

Primary Aims : Assessment of the quality of life post surgery with a disease specific questionnaire and to assess the degree of improvement

UGI scopy findings pre and post surgery

Secondary Aims : To see if any patient is still having to rely on PPI's

Any complications post surgery(if any)

Justification of the study : To assess whether Laparascopic fundoplication really improves the quality of life compared to medical manaement and to assess the usefulness of surgery based on risk vs benefit ratio

MATERIALS

A Prospective / Retrospective study conducted in PSG Institute of Medical Sciences and Research to assess whether Laparoscopic fundoplication really improves the quality of life compared to medical management and to assess the usefulness of surgery based on risk vs benefit ratio by comparison of their preoperative and postoperative Quality of life with a questionnaire and endoscopic findings in 31 patients

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ANTIREFLUX MEDICATION (1 point for each)

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- Antacids
- Ca channel blockers
- Anti anxiety drugs
- Prokinetics

Type of Study

Observational, Prospective and Retrospective

Inclusion Criteria

Clinical reflux symptoms

UGI endoscopy

Exclusion Criteria

H/o previous oesophageal surgery

Complicated reflux (eg. Ulcer, Short oesophagus)

METHODOLOGY

In this study 30 patients who had clinical symptoms of gastro oesophageal reflux were included.

Informed consent was obtained detailing the study process, need for the study and its benefits, elaborately. The history was recorded by the principal investigator.

The questionnaire was filled diligently by the principal investigator.

All 31 patients were subjected to clinical examination by the principal investigator in an unbiased manner.

Their Upper GI endoscopy findings were recorded, along with Oesophageal manometry and USG abdomen as and when required. Clinical symptoms of

patients, especially those refractory to long term medical management and findings with UGI scopy played a role in patient selection for surgery.

After the patients underwent laparoscopic fundoplication, re evaluation of their symptoms and their quality of life was done with History, clinical examination and questionnaire and repeat UGI scopy.

RESULTS

Analysis of Data:

A total of 31 cases were included in the study

1 patient did not present for follow up

For 30 cases informed written consent was obtained

For all 30 cases preoperative and post operative history was elicited and questionnaire completed

For 31 cases preoperative UGI scopy done

For 5 cases preoperative Oesophageal manometry was done

For 28 patients post operative USG was done. (2 patients were not willing for follow up USG)

Pre operatively all 31 patients were taking either PPI's, Antacids, Prokinetics or the combination of above. Post operatively 16 patients were still taking PPI's. (It must be noted that there was marked relief of symptoms, with medication post Surgery compared to prior to surgery, in which a majority of patients were refractory to medical management). This compares favourably with the international standard of patients on pre and post surgical medication)

According to the Health Related Quality of Life questionnaire, the relief of symptoms post surgical management is 93% which again compares favourably with international standards

According to UGI scopy findings

10 subjects had Grade 1 reflux oesophagitis post surgery

3 patients among them had antral gastritis

1 patient had erosive gastritis

3 patients had prolapse gastropathy

1 patient had a small hiatal hernia post surgery

Rest of 27 patients had intact fundoplication wrap

These recurrence rates are on par or better in comparison with international standards

DISCUSSION

A prospective / retrospective study of evaluation of 30 patients undergoing laparoscopic nissens fundoplication in PSG hospitals was done and it infers the following.

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